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CONTENTS OF PREVIOUS NUMBER

JULY 1948. NUMBER 1

Herpes Zoster Oticus ("Ramsay Hunt Syndrome"): Report of a Case. Leighton Johnson, M.D., and Bernard Zonderman, M.D., Boston.

Cleft Lip and Palate: Some Technical Procedures Used in Its Treatment, with Especial Reference to Closure of Complete Cleft of the Anterior Half of the Palate. Fred Z. Havens, M.D., Rochester, Minn.

The Middle Ear in Sound Conduction. Ernest Glen Wever, Merle Lawrence and Kendon R. Smith, Princeton, N. J.

Thrombophlebitis of the Cavernous Sinus Originating from Acute Dental Infection: Cure with Penicillin. Kenneth S. Oliver, M.D.; Alfred E. Diab, M.D., and Caesar N. Abu-Jaudeh, M.D., Beirut, Lebanese Republic.

Cigaret Smoke Irritation: III. The Efficiency of Filters in Reducing Its Intensity. H. B. Haag, M.D.; J. K. Finnegan, Ph.D., and P. S. Larson, Ph.D., Richmond, Va.

Incidence of Congestion of the Larynx in Men and in Women. Gerard H. Cox, M.D., Saranac Lake, N. Y.

Auditory Nerve in Multiple Sclerosis. Hans Von Leden, M.D., and Bayard T. Horton, M.D., Rochester, Minn.

Bartolommeo Eustachio: A Great Medical Genius Whose Chief Masterpiece Remained Hidden for One Hundred and Fifty Years. Walter A. Wells, M.D., Washington, D. C.

p_H of Nasal Secretion in Situ in Infants and in Children: The Effect of Hyperventilation and Crying. Noah D. Fabricant, M.D., and M. A. Perlstein, M.D., Chicago.

Rhinoplasty in Children, with a Report of an Operation on a Nine Year Old Boy. Irving M. Lefkon, M.D., New York.

Case Reports:

Total Stenosis of the Esophagus Due to Lye Burn. Safa Karatay, Ankara, Turkey.

Progress in Otolaryngology:

Summaries of the Bibliographic Material Available in the Field of Otolaryngology: Contributions to Plastic Surgery During 1946. Lyndon A. Peer, M.D., and John Van Duyn, M.D., Newark, N. J.

Abstracts from Current Literature.

News and Comment.

Directory of Otolaryngologic Societies.

ARCHIVES OF OTOLARYNGOLOGY

VOLUME 48 AUGUST 1948 NUMBER 2

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BACTERIAL ALLERGY IN RELATION TO ASTHMA

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CHICAGO

BECAUSE of an increasing interest in the management of patients having allergic respiratory symptoms, it might be of value to review the observations made on a group of persons with this rather common disability and to outline a method of treatment which has given satisfactory results. This group of 100 patients included 36 adults and 16 children, all of whom were known to be asthmatic, and 48 children with allergic bronchitis or allergic rhinitis or both; the last-named group could doubtless be classified as preasthmatic. Gray and Albert [1] designated as preasthmatic symptoms nasal congestion, sneezing, rhinorrhea, cough and dyspnea and emphasized the importance of taking every possible precaution with a potentially asthmatic person to prevent the first actual asthmatic attack.

Early in our study of infections of the pharynx, especially the nasopharynx, my associates and I were impressed with the number of allergic patients who gave a history of infections of the respiratory tract related to allergic attacks and who showed clinical and bacteriologic evidence of active or latent nasopharyngitis. Frequently the first, and often the second, local treatment precipitated a recurrence of symptoms, this event seeming to indicate a bacterial factor. We were also impressed by the fact that in a number of cases symptoms subsided after adequate treatment of the infected area.

From our work with filtrate in local immunization in combating other types of diseases of the respiratory tract, there developed this combined treatment for asthma, allergic bronchitis and allergic rhinitis. It is directed toward clearing up the infection in the pharynx by local therapy, followed by the use of bacterial filtrate as nose drops. First a swab moistened with an aqueous solution of benzalkonium chloride ("zephiran chloride") is used to remove the film of mucus which usually covers the membrane of an infected nasopharynx. Then an

† Dr. O'Connor died on March 25, 1948.

1. Gray, I., and Albert, M. M.: Asthma: Prevention in Industry, Indust. Med. **12**:801-804 (Dec.) 1943.

astringent (3 to 10 per cent solution of silver nitrate) is applied. The third, and last, application is with a swab moistened with a solution containing 50 per cent acriflavine, 25 per cent methyl violet 2 B and 25 per cent crystal violet in 1 per cent concentration. If there is no response after three or four treatments, a weak solution of iodine is used instead of silver nitrate, and, in addition to the application of the acriflavine mixture, bacterial filtrate, in a base of vanishing cream, is applied. Usually, after the second treatment in children and the sixth in adults, the patient is given a bacterial filtrate and instructed to use 4 drops in one nostril twice a week.

Filtrates are prepared in the following manner: Material for culture is obtained from the throats of patients having acute and chronic pharyngeal infections. Blood agar plates are streaked with the material and incubated for at least forty-eight hours. When an isolated colony of the desired organism can be selected directly from the original culture, it is planted in the medium for filtrate preparation at the same time that subcultures are made for verification of identification and purity of culture. If the original culture is too heavy, isolation must be made, of course, before transplantation to the filtrate medium. A flask containing 25 to 50 cc. of dextrose-beef infusion broth is inoculated, incubated for from five to eight days and the contents filtered through a Berkefeld filter. The filtrate is tested for sterility and is then preserved with "merthiolate," 1: 5,000. Filtrates for each of the following organisms are prepared: Streptococcus viridans, hemolytic and nonhemolytic streptococci, the pneumococcus and staphylococcus, Neisseria catarrhalis and Hemophilus influenzae. In preparation of the filtrate for the last-named organism, a few drops of human blood are added to the broth in which the cultures are grown. Many filtrates are pooled, to make up the polyvalent stock filtrate for each species.

The filtrate given the patient is made from these stock polyvalent preparations, diluted 1: 36,000 with a sterile isotonic solution of sodium chloride. The species which are included depend on the culture obtained in the individual case. Occasionally, in the case of a patient who shows no response to the stock filtrate, an autogenous preparation is combined with the stock solution. If an asthmatic attack is precipitated by the use of the filtrate, the dilution is increased, the degree of dilution depending on the severity of the reaction. The reaction, if any, usually occurs within thirty-six to forty-eight hours.

We have observed that the filtrate is more effective if one or more local treatments are given to decrease the congestion in the nasopharynx usually found in these cases. In children, use of the filtrate may be started after the first or second treatment, and in many cases no

further local treatment is necessary. If the use of the filtrate is persisted in for a three year period, the tendency to recurrence is slight. In adults, it is necessary for a series of treatments to be given before the filtrate is started and, in a few cases, an occasional treatment thereafter. In case of recurrence, one or two treatments usually suffice to relieve the symptoms.

Of the group of 100 patients included in this review, 22 had been under the care of an allergist and had been given the usual skin tests and desensitizing treatment, without improvement. Allergists, as well as rhinologists, seem agreed that infections of the upper respiratory tract almost invariably intensify allergic symptoms, even in patients who have a recognized sensitivity to a particular food, pollen or other nonbacterial allergen. In other words, while the skin tests give definitely positive reactions for certain foods or pollens, the absorption of bacterial toxin seems to be the trigger that sets off the allergic attack. Of interest in this connection is the observation on 3 children who had definite food sensitivities at the beginning of treatment and who were able to tolerate all foods as soon as the infection was controlled.

At the time of the patient's first visit to the office, a complete examination of the ears, nose and throat is made. A nasopharyngoscope is passed routinely if the patient is old enough to cooperate. This procedure is simplified by the instillation of a few drops of a 2 to 4 per cent solution of procaine borate into the nose, which measure produces an immediate, though brief, anesthesia. Material for cultures is obtained from various parts of the pharynx. All patients in the present series showed evidence of infection of the pharynx of varying degree. Fifty-three per cent of the asthmatic patients stated voluntarily that their attacks had been precipitated by or had followed an acute infection of the respiratory tract. Sixty-nine per cent had had their tonsils removed previously; 55 per cent of the allergic children were without tonsils. None of the patients had an aural infection. No cases of purulent infection of the sinuses or of polypi were included in the series.

REPORT OF CASES

Case 1.—Illustrative of the asthmatic group is the case of F. W., who was first seen by us in August 1939, at the age of 19. He gave a history of asthma since the age of 6, for which he had been under constant treatment by an allergist. The attacks had been most frequent and severest from August to October, in extreme cold weather and after an acute infection of the respiratory tract. His physical activities were necessarily drastically curtailed. Our examination revealed decided congestion of the mucous membranes of the nose, nasopharynx and oropharynx, and cultures from the nasopharyngeal area showed a moderate growth of Str. viridans. Local treatments, supplemented with nose drops of autogenous filtrate, were given for several weeks, with great improvement in symptoms. In June of the following year, after an acute infection of the upper

respiratory tract, there was recurrence of the asthmatic attacks, and at this time the pneumococcus was found on culture. The response to local treatment was again satisfactory, and no further attacks occurred during the winter. The patient continued to use the filtrate and had no recurrences, was accepted in the Army Air Corps and served overseas. His condition when we first saw him in 1939, would certainly have precluded his acceptance in that branch of the service. Since his return to civilian life he has had no recurrence of asthmatic attacks.

CASE 2.—J. W., a boy aged 10, with a history of asthma of six years' duration, was first seen by us in May 1942. During the previous summer, while under the care of an allergist, he had shown positive reactions to one hundred skin tests, especially to those with wool and feathers. Just before he came under our care, a number of food tests had been made, but none of the foods to which he had shown sensitivity were eliminated during our course of treatment.

The examination of the nose and sinuses showed an essentially normal condition. The nasopharynx and oropharynx were inflamed; cultures from the former produced a heavy growth of hemolytic streptococci and a moderate growth of N. catarrhalis, Staphylococcus aureus and nonhemolytic streptococci; cultures from the oropharynx showed a moderate growth of N. catarrhalis and Streptococcus hemolyticus and a few colonies of Str. viridans.

The patient, although young, was given a series of treatments because of the long history, the type of infection disclosed by the cultures and the intense congestion of the pharynx. After the third treatment he was given a stock filtrate, to be used as nose drops. Improvement was gradual throughout the summer, and in September the cultures were practically sterile. The patient was instructed to continue to use the filtrate, and he remained well throughout the winter. Asthma recurred to some degree after a cold in November 1943, at which time the patient stated that he had not used the filtrate during the summer. Since resuming its use, he has had no asthmatic attacks. There has been no recurrence of food sensitivity.

CASE 3.—B. B., aged 5 years when seen in March 1941, is representative of the group of allergic children. He was brought to us with symptoms of a continuous cold and sneezing. A roentgenogram of the sinuses had shown them to be normal, and a series of roentgen treatments had afforded no relief of symptoms. Our examination revealed no infection of the sinuses. The membranes of the nose appeared turgescent, and the nasopharynx was inflamed and congested. After one local treatment of the nasopharynx, the patient was instructed to use the filtrate as nose drops. When he was brought in six months later for further examination, the mucous membranes appeared normal, and the parents reported that he had had no symptoms since the latter part of March. He continued to use the filtrate for two years and has had no recurrence of the allergic symptoms.

Of the 52 asthmatic patients, 19 (36.6 per cent) obtained complete relief and have been entirely free from symptoms for periods ranging from one to six years. Usually six local treatments were given; in a few cases a longer series was necessary. Twenty-nine (55.7 per cent) have had the asthma brought under control for the most part but have had occasional recurrence following an acute infection of the respiratory tract. Of the asthmatic group, then, the results were good or fair for 92.3 per cent. Four patients have not been helped appreciably by this or any other type of treatment for asthma which they have received.

The record of results with the group of 48 allergic children is less complete, since in many instances the patient was seen only once and was given filtrate to use as nose drops at home. Of 32 patients in this group who have been followed for years, 14 (47 per cent) have been completely free from allergic symptoms. Twelve (37.5 per cent) have experienced no trouble except occasionally after a cold, and then only for a short time. Of the 32 allergic children who have been followed, results have been good or fair in 84.5 per cent. The remaining 5 patients have shown only slight improvement. On the whole, therefore, the results seem to justify further use of this method of treatment.

SUMMARY

Infection and bacterial allergy are being recognized as important factors in asthma and other allergies referable to the respiratory tract.

A twofold treatment is described. The first step is directed toward eradication of the infection in the pharynx by cleansing the area of the film of mucus and applying an astringent, followed by the application of bactericidal agents. The second step consists in the local use of bacterial filtrate, in an attempt to stimulate the body's own protective mechanism.

This study is based on a group of 100 patients having allergic symptoms referable to the respiratory tract. Three illustrative cases are discussed.

The results with the asthmatic patients were good or fair in 92.3 per cent. Of the allergic children who reported for follow-up observation, the results were good or fair in 84.5 per cent.

30 North Michigan Avenue (2).

SPEECH THERAPY

Experiences with Patients Who Had Undergone Total Laryngectomy

ESTI D. FREUD
NEW YORK

RETRAINING the voice of the totally laryngectomized patient affords great satisfaction to the speech therapeutist. The Speech Clinic of the Manhattan Eye, Ear and Throat Hospital has retrained over 50 such patients in the past four years. They have been taught to substitute for the lost laryngeal voice sounds which are possibly produced in the esophagus, and to use these sounds for articulated speech.

The psychologic trauma from loss of voice may be as great as the physical shock of the laryngectomy. The frustration of not being able to communicate with others may become a serious impediment to voice recovery. Therefore, the policy of the clinic is now to begin speech training immediately after the removal of the patient's feeding tube. The training of 4 patients was begun even prior to the laryngectomy, with the expectation of better and speedier results.

Four years ago, at the inception of this work, my associates and I had to use all our persuasion to convince the patients to try speech rehabilitation. They first came to their lessons with more or less reluctance and apprehension.

Some of these patients, especially the younger ones, were so upset by their infirmity, that they simply lost any desire to talk and to associate with other human beings. They had the attitude of the child who said: "It serves my parents right that my fingers are frozen; why don't they buy me new gloves?" The older men, those over 65, did not want to be bothered any more. Their attitude was: "What's the use of making so much effort!" All the patients were suspicious and doubted that good speech would result.

Our patients were distributed in age groups as follows:

	39-40	40-45	45-50	50-55	55-60	60-65	65-70
Men	1	0	6	11	16	14	5
Women	-	-	-	1	1	-	-

From the Speech Clinic of the Manhattan Eye, Ear and Throat Hospital, service of Daniel S. Cunning, M.D., surgeon director.

That the age group from 65 to 70 is so poorly represented among them is a proof of our statement that the older patient lacks the energy necessary to acquire the audible esophageal voice.

Fortunately, this attitude has completely changed in the last two years. A sufficient group of patients whose pseudovoices are excellent is now at our disposal. Each of these patients has to pay regular visits to the hospital for follow-up examinations; on this occasion the patient goes to the ward and gives a "pep talk" to the patients newly operated on. It is amazing how this new arrangement has improved the morale of the postoperative patient. Now there is hope instead of discouragement, and instead of low spirits, confidence and ambition to speak again, "as well as Sam, better than Sam." This kind of procedure vigorously stimulates the patient's wish for rehabilitation and for resumption of a normal life with normal relationships.

A few weeks after the operation all the patients produce a whispered speech, using for articulation the air accumulated in the mouth. We have heard patients trying to speak in a kind of snore language, with sounds that were probably formed between the upper pharyngeal wall and the velum. The loudest and therefore the best voice substitute is the so-called esophageal voice.

Speech is composed of two functions: phonation (or voice production) and articulation. The total laryngectomy has in no way damaged the patient's organs of articulation but has completely removed his organ of phonation. Normally human voice is a loud expiration. Breathing may be transformed into voice whenever the motor speech center, through the specific nerve conduit, orders the vocal cords to close, so that the passing breath current may bring them into vibration; or, in other language, breathing may be transformed into voice whenever one wishes to speak. The correlation between breathing and voice is half conscious in a layman but is clearly conscious and trained in any vocal artist, such as a singer, an actor or a clergyman.

In the following passage an effort has been made to describe the method which in our experience has been most efficacious in obtaining early results. The first and most important fact that the patient has to learn is to dissociate breathing from the function of speech. A new pattern has to be created to take over phonation.

As psychologic factors play a predominant role in this therapy—as in all voice and speech treatments—we always try to produce the esophageal voice at our first session with the new patient. To insure the occurrence of this phenomenon, we administer half a teaspoon of sodium bicarbonate in a cup of water to the patient, advising him to swallow some air on top of it; he is further told to fix his mouth in an "A" position ("A" as in garden) in order to be ready to articulate simultaneously with the eructation. Only 1 of 3 patients immediately

succeeds in synchronizing and coordinating the ructus with the enunciation of the vowel. In most cases the belch escapes and is followed by a belated attempt at articulation. These efforts of the patient to apply the old breath-voice pattern result in irritation of the trachea and in coughing spells. Whenever this happens, it is advisable to postpone further training until the next day. Again and again it has to be explained and emphasized that the new voice is to be completely dissociated from exhalation and that the patient has to avoid up and down movements of the thoracic cage during the pseudophonation; instead he has to concentrate on contracting the diaphragm.

Another difficulty is the air supply for more than one syllable. The practice of administering sodium bicarbonate is abandoned as soon as it has produced its psychologic effect, and from now on the patient is taught to swallow air and to reject it.

Usually after four or five lessons he manages to store sufficient air for saying two or three syllables without interruption. From now on

Fig. 1.—Roentgenogram of the stomach and a diagram of the air volume of the stomach as determined from the size of the gastric vesicle: (*a*) normal; (*b*) enlarged by swallowed air; (*c*) reduced by phonation.

it is only a matter of time, which may vary from a few weeks to six months or more, until the patient gets accustomed to accumulating in the stomach, in the esophagus or in the hypopharynx the air necessary for the fluency of understandable speech. For the strengthening of the pseudovoice, exercises with prolonged vowels, such as oooooooooo or eeeeeeeeeee, prove effective.

The results of our speech rehabilitation may be tabulated:

	Excellent	Good	Fair	Abortive
Men	9	29	11	4
Women	1			1

As to the opinions concerning the location of this air vesicle, they are of remarkable variety. Hajek [1] placed it in the stomach and showed

1. Hajek, M.: Pathologie und Therapie der Erkrankungen des Kehlkopfes, der Luftröhre und der Branchien, Leipzig, Verlag K. Kabitzsch, 1932.

a roentgenogram and a diagram which demonstrated the stomach enlarged by air before pseudophonation. Stern [2] expressed the opinion that the air reservoir changes place during treatment (this is also our opinion), beginning in the stomach and ascending later to the esophagus and the hypopharynx. The Pancoast-Pendergrass-Schaeffer [3] roentgenograms of the neck of a patient who had undergone total

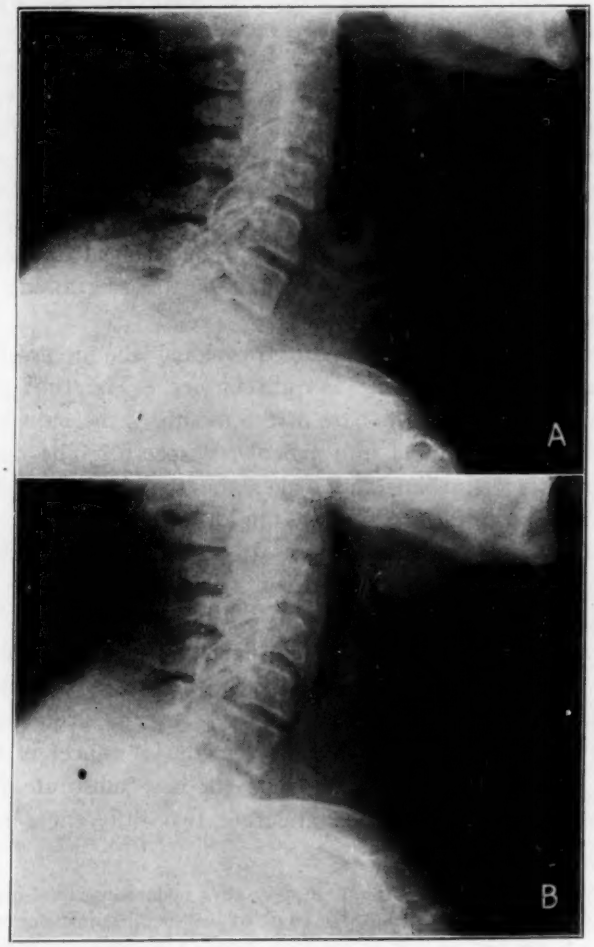


Fig. 2.—Roentgenograms of the hypopharynx taken during phonation: *A*, before phonation; *B*, after phonation.

2. Stern, H., in Gutzmann, H.: Sprachheilkunde: Vorlesungen über die Störungen der Sprache mit besonderer Berücksichtigung der Therapie, Berlin, H. Kornfeld, 1924.

3. Pancoast, H. K.; Pendergrass, E. P., and Schaeffer, J. P.: The Head and the Neck in Roentgen Diagnosis, Springfield, Ill., Charles C Thomas, Publisher, 1940.

laryngectomy, taken during phonation, showed accumulation of air in the upper part of the esophagus. Roentgenograms which Dr. W. R. Cashion, radiologist to the Manhattan Eye, Ear and Throat Hospital, made of one of our patients during phonation present modification of the air volume of the hypopharynx. We were not able to observe any change in the size of the stomach.

To those patients who were unable to learn the esophageal voice and to those who refused training we recommended selecting one of the two models of an artificial larynx, the reed type larynx of the Bell Telephone Laboratories or the Wright "electro-larynx." These models are far from being a satisfactory solution of the problem. (With use of the Bell larynx, the tube containing the reed is inserted in the trachea; thus it uses the breath current for sounds. This tube is connected at right angles with another tube ending in a mouth piece, which consists of a rubber membrane for articulation.) The tracheal tube of the Bell larynx may become a dangerous irritant of the scar tissue and may produce coughing spells.

An electric battery and a motor providing the necessary sounds are the basic elements of the Wright larynx. The tube containing the soundmotor is put on the base of the mouth of the patient, and the sounds are thus transmuted into articulated speech. The noise of the electric motor is disturbing and makes the speech difficult to understand. Both machines produce an unpleasant, artificial, monotonous type of speech and are conspicuous. It seems to us that their worst disadvantage is that one of the patient's hands is constantly occupied with manipulation.

People undergo operations to free themselves from the necessity of wearing a hearing aid. The necessity of wearing a complicated and embarrassing voice apparatus can be overcome by acquiring a good esophageal voice. As long as the only remedy for cancer of the larynx is removal, a good pseudovoice is by far the best substitute.

The following case histories illustrate two different rehabilitation problems.

S. J., 51 years old, left the hospital ten days after undergoing total laryngectomy. His recovery had been uneventful. He came to the speech clinic one month later. When we first saw him he was in excellent health but extremely depressed. He badly needed speech for his business. We administered sodium bicarbonate and explained the procedure. With the first eructation the patient could say his name. He became so excited when he again heard his voice that tears filled his eyes. After ten minutes of highly dramatic events this patient had mastered the new technic and completely recovered his faculty of speech. He still is one of our most gifted pseudovoice patients.

Fred M. was 39 years old when he underwent total laryngectomy. He began speech training while still in the hospital, immediately after the removal of the feeding tube. Although he learned to belch without sodium bicarbonate, he was

not able to associate articulation with the ructus. All our attempts to explain the new correlation failed and were accompanied by the skeptical smile of Mrs. Fred M., who insisted on being present during the training. After six weeks of fruitless efforts the patient's wife told us that he had to stop coming to our clinic as she could not afford to pay any longer for the transportation. Four months later the patient suddenly reappeared with a good although not strong pseudovoice. He told us that he had practiced alone until he discovered the knack of it. He is gainfully employed, and he comes to our clinic once a month on his day off for voice strengthening.

TRACHEOTOMY IN POLIOMYELITIS SIMPLIFIED WITH NEW RESPIRATOR

R. L. PETERSON, M.D.
AND
R. C. WARD, M.D.
BOISE, IDAHO

THE life-saving effect of tracheotomy in certain types of poliomyelitis was first reported by Galloway in 1943.[1] His work has been corroborated in several epidemics since that time.[2] In the Idaho epidemic of 1947 approximately 400 poliomyelitis patients were hospitalized in St. Luke's Hospital in Boise. It is our opinion, from our experiences in this epidemic, that tracheotomy when properly indicated has been a life-saving measure. Galloway's recent recommendations[3] as to indications and technic have been followed as closely as possible.

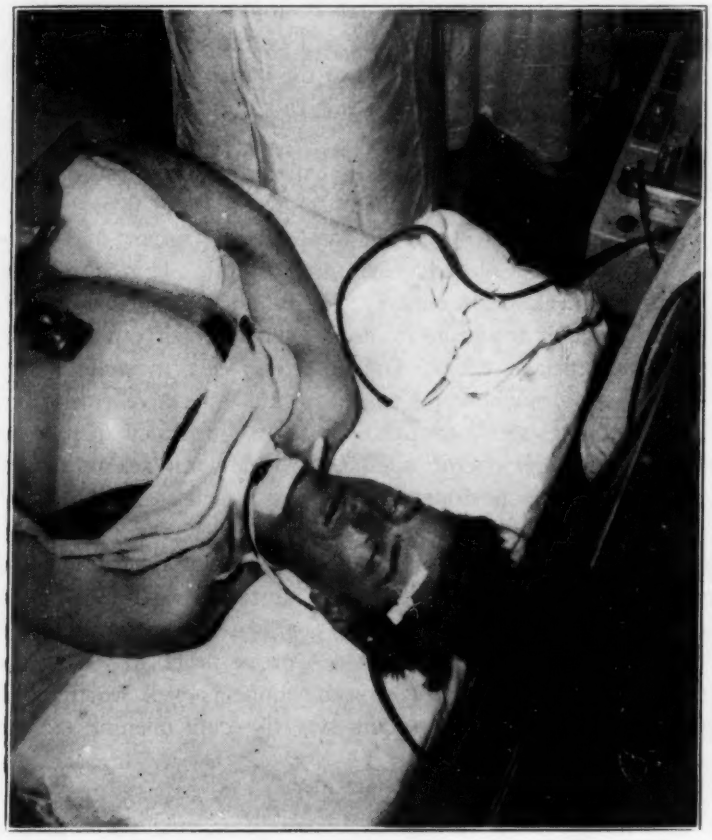
Tracheotomies performed on patients that were not in respirators have presented no particular problem. In these cases an airway has been established by means of a bronchoscope or an endotracheal tube, The anesthetist has taken charge of the airway and proper oxygen flow, and the laryngologist has done a well planned and "orderly" tracheotomy. The postoperative care of these patients has presented no special problem. However, patients with severe respiratory paralysis who had to remain in respirators have been much more difficult to handle. The technic followed has been first to establish an airway and proper oxygen flow, then to open the respirator and do a tracheotomy with the respirator open, artificial respiration being given if necessary. In our experience this procedure has been carried out successfully, but usually with considerable difficulty and confusion. It has likewise been reported by others that this procedure has often been accompanied with considerable difficulty and has added a definite and important element of anoxemia and cardiac strain.[3] With this technic, time is important, and an orderly tracheotomy can seldom be done. On one occasion, our

1. Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. **123**:1096 (Dec. 25) 1943.

2. Rocky Mountain Conference on Infantile Paralysis, sponsored by the University of Colorado School of Medicine and Hospitals and the National Foundation for Infantile Paralysis, Inc., December 1946.

3. Galloway, T. C.: Management of Respiratory Complications of Poliomyelitis, Arch. Otolaryng. **46**:125 (Aug.) 1947.

patient became seriously anoxemic before the operation was completed and the respirator closed. Prophylactic tracheotomy has been advocated for cases of bulbar poliomyelitis in which the patient is seen at an early stage, to prevent the necessity of carrying out this difficult procedure on a patient in the respirator.[3] Many times this precautionary measure is not possible. Most of our patients with the bulbar type of



A recently tracheotomized patient with severe bulbar poliomyelitis showing new type, collarless respirator.

the disease have come from outlying rural districts, and because of this and other unpreventable circumstances, the patient has not been seen early.

After the tracheotomy, no little difficulty has been encountered with the collar of the tank type respirator because it interferes with proper function and care of the tracheotomy tube. This difficulty has been partially eliminated by a special neck piece for the respirator to which a wire frame is attached that holds the collar away from the tracheotomy

tube and allows more room for proper care of the tube and aspiration of its content. We have found that with this special neck piece and frame there has still been considerable difficulty in the postoperative care. Changing of the inner cannula is difficult. Slight changes in the position of the patient often cause difficulty, and unless the attendant is especially deft, the tube is not handled in the gentle manner it should be. On one occasion, in one of our cases, the wire frame holding the collar became misplaced, and the tracheotomy tube was forced out of the wound by the respirator collar. The nurse in attendance could not replace the tube without help, and the patient suffered considerable respiratory embarrassment before the tube was replaced and proper respiration restored.

The Blanchard and Mullikan so-called lung respirators have not been practical for tracheotomy because of the tight-fitting collar. A new respirator has been developed by J. J. Monaghan, of Denver,[4] in cooperation with the Colorado General Hospital. This respirator does away with the neck piece entirely. It in no way interferes with the technic of a regular tracheotomy done over a properly placed bronchoscope or endotracheal tube. The head is freely movable for proper insertion of the bronchoscope or tube. Postoperative care is greatly simplified. It has been our privilege to use this new respirator. It greatly simplifies the operation of tracheotomy on respirator patients and greatly simplifies postoperative care.

In our limited experience this respirator has been adequate to sustain respiration postoperatively. If desired, the patient can be returned to the tank respirator. We have had no difficulty and the patients have suffered no ill effect in being transferred from one respirator to the other. Should it not prove practical for long usage, we feel its being used for tracheotomy alone is well worth while and affords a tremendous improvement over the tank opening technic.

SUMMARY

Tracheotomy, properly indicated, is a life-saving measure in poliomyelitis. Previous difficulties encountered in doing tracheotomy on a patient in the conventional type of respirator can be overcome by the use of the new type, collarless respirator. Operative technic and postoperative care are greatly simplified.

218 Eastman Building.

4. The new type respirator is marketed by J. J. Monaghan & Company, Denver.

EFFECT OF PREGNANCY ON OTOSCLEROSIS

HAL WAUGH SMITH, M.D.
NEW YORK

SINCE Lempert's [1] original article appeared, in July 1938, describing a one stage operation that gave surgical relief from deafness in a majority of cases, a new interest has been initiated in regard to the problem of otosclerosis. While most of the interest has been in the field of improving the results of the surgical technic, this new focus of attention on the disease has also uncovered many adjacent fields of study. Not the least of these is that of the relationship of pregnancy to this type of hearing loss.

Nager's [2] observations are almost classic. He studied the histories of 1,146 cases of otosclerosis and many sections of temporal bones. Some of his findings were:

1. Otosclerosis occurs in 10 per cent of all adults (histologically), but only a small portion of these show it symptomatically (poor hearing).
2. The sex ratio of clinical otosclerosis is 1 male to 1.8 females.
3. In over half the cases the clinical onset occurs between the ages of 16 and 30 years.
4. Hereditary or familial deafness was found in over 50 per cent of his cases.
5. Of 383 married women with otosclerosis, 80 showed an increase of deafness with pregnancy.

One notes in the foregoing summary that 80 of the 383 married women with otosclerosis showed an increase of deafness from pregnancy. That is a ratio of 1 in 4.8, or 20.8 per cent.

Shambaugh,[3] reviewing Nager's [2] article, agreed with him. In his experience, pregnancy has resulted in a hearing loss for 1 patient in 5, or 20 per cent.

From the New York Eye and Ear Infirmary.

1. Lempert, J.: Improvement in Hearing in Cases of Otosclerosis: New One Stage Surgical Technic, Arch. Otolaryng. **28**:42 (July) 1938.

2. Nager, F. R.: Clinical Manifestations and Pathologic Anatomy of Otosclerosis, Nord. med. (Hygiea) **2**:1703 (June 3) 1939.

3. Shambaugh, G. E., Jr.: Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear, Arch. Otolaryng. **33**:436 (March) 1941.

Searching in the literature, one finds varying degrees of incidence reported. Guggenheim [4] expressed the belief that over 50 per cent of women with otosclerosis lose in hearing during or after pregnancy. On the other hand, Bunch [5] tested 6 women having clinical otosclerosis, making careful audiometric studies both before and after pregnancy. In none of the 6 women was there a loss of hearing because of pregnancy.

Brunner,[6] in a general article on otosclerosis, presented 3 cases that illustrate the paradox in the linking of this disease to pregnancy.

His first case is one of a woman who at autopsy had definite pathologic evidence of otosclerosis in her right ear. Prior to her death she had shown no clinical hearing defect and had given birth to four children.

His second case is also one in which at autopsy otosclerosis was observed—in this instance, bilateral. Prior to death the patient had experienced a serious loss of aural function; however, her history revealed that she had never been pregnant.

His third case is most interesting. The patient underwent bilateral fenestration, with partial restoration of hearing on one side. Several months later her first child was born. This did not affect her hearing. Two years later a second child was delivered. This time the patient felt she had suffered a slight loss in aural acuity. An audiogram, however, proved to be almost identical with the one made after fenestration. A year later the patient became pregnant for the third time. In the fourth month of gestation an audiometric examination showed what she had subjectively noted: a slight improvement in hearing! Shortly thereafter the patient spontaneously aborted. An audiogram made after she had recovered from this mishap was found to be essentially the same as the previous one!

Otosclerosis appearing in identical twins has caught the attention of investigators because it offers an opportunity to follow the clinical appearance of the disease in 2 patients of a similar hereditary background. For the present purpose, one is primarily interested in the effect of pregnancy in such patients.

Hagens [7] has reported an instance of otosclerosis occurring in identical twins. The onset of poor aural acuity in one of the pair occurred at the age of 17. In the other the onset was a few years later. The twin with the earlier onset became pregnant a year later. This resulted

4. Guggenheim, L. K.: On the Diagnosis and Treatment of Otosclerosis, Am. J. Surg. **43**:156 (Oct.) 1938.

5. Bunch, C. C.: The Progress of Deafness in Otosclerosis, Laryngoscope **49**:793 (Sept.) 1939.

6. Brunner, H.: Otosclerosis, Laryngoscope **53**:736 (Nov.) 1943.

7. Hagens, E. W.: Otosclerosis in Identical Twins, Arch. Otolaryng. **34**:583 (Sept.) 1941.

in a decided increase of hearing loss. The twin with the later onset of symptoms had never been pregnant, and her defect was the lesser of the two.

Another paper on this same topic was written by Fowler.[8] He presented detailed observations on three pairs of identical twins. If his case histories are surveyed closely, one finds that it was only from the third pair that a positive familial history of deafness was elicited. A maternal grandfather reported that two of his sisters attributed their deafness to childbirth. The older sister, with but one child, suffered almost immediate loss after delivery. The other, with three children, had a loss in three progressive steps with the three successive children. In contrast with this, the mothers of the three pairs of twins had no hearing defect, and the delivery of the twins was accomplished without difficulty. Furthermore, one girl twin of this group of six had married and given birth to two children without further impairment of her defective hearing.

The literature since July 1938, which I have reviewed, includes two papers that attempt to deal solely with this problem of otosclerosis and pregnancy.

The first of these was by Allen.[9] It was read at the Sixty-Eighth Annual Meeting of the American Gynecological Society in 1944. In his paper he presented statistics on 72 cases of clinical otosclerosis in which there had been one or more pregnancies. Of his 72 cases, 35 were taken from the literature, 32 were from personal communications and 5 from among personally observed cases.

He reported the incidence of hearing loss associated with pregnancy as 35 of 72, or 48.6 per cent. In 14 of these instances, 19.4 per cent, the loss occurred with the first pregnancy. In the remaining 21, 29.1 per cent, it occurred with pregnancies other than the first.

He concluded that otosclerosis is not an indication for therapeutic abortion in the primipara; nor is it a contraindication for marriage and a trial pregnancy. He stated, though, that a multiparous woman may, if accurate evidence from previous pregnancies shows a definite sustained loss of hearing, rightfully question further pregnancies. Before conceding that an interruption is in order, he recommended that the diagnosis of otosclerosis be definitely established; a familial history, while not required, is to be preferred; also, the patient should be informed that the disease is a progressive one even without pregnancy and that lip reading and the hearing aids may help.

8. Fowler, E. P.: Studies of Deafness in Twins: Otosclerosis in Identical Twins; Three Case Histories, Laryngoscope **52**:718 (Sept.) 1942.

9. Allen, E. D.: Pregnancy and Otosclerosis, Am. J. Obst. & Gynec. **49**:32 (Jan.) 1945.

The other paper represents Barton's [10] findings in a survey at the Massachusetts Eye and Ear Infirmary.

He gathered data on 133 otosclerotic female patients for analysis. The hearing loss in 73, 55 per cent, of these women was either increased or precipitated by pregnancy. In 51, 38.3 per cent, the impairment occurred as the result of the first pregnancy. In 22, 16.5 per cent, it occurred with the second or subsequent pregnancies. Of multiparous women who suffered a loss of hearing with their first offspring, about 50 per cent showed no further loss with further gestations.

He presented 2 cases in which therapeutic abortion was performed. The first patient's hearing difficulty followed delivery of her first child and was increased with delivery of her second. When she became pregnant a third time, abortion and sterilization were done in the second month of intrauterine life. No change of hearing function was noted after this third pregnancy or the surgical intervention. In the other case the hearing defect had begun before any period of gestation. During her first pregnancy there was no change in hearing acuity, but her tinnitus was increased. On this basis a therapeutic abortion was performed during the third month. Later, this patient again became pregnant and was permitted to go to term and deliver. She did so without any ill effects on her hearing.

He presented 1 case in which, as in Brunner's [6] third case, an improvement of hearing occurred during pregnancy. After the postpartum period was completed, the patient's hearing resumed the path of slow loss.

The author concluded that since the effect of pregnancy on otosclerosis is variable and unpredictable abortion is not justified; further, that sterilization is futile in controlling the disease, as it is impossible to prophesy deafness of the offspring.

The sterilization of otosclerotic mothers, including the surgical interruption of any current pregnancy, as a means of prophylaxis has more active proponents. Greifenstein,[11] collecting statistics from a number of European clinics, found 79 cases in which it was performed. He was of the opinion that auditory disturbances were not increased as much by the first pregnancy as by later ones.

In this country, Guggenheim,[12] writing on the prophylaxis of otosclerosis, recommended early diagnosis so that (1) two otosclerotic

10. Barton, R. T.: The Influence of Pregnancy on Otosclerosis, New England J. Med. **233**:433 (Oct. 11) 1945.

11. Greifenstein: Interruption of Pregnancy and Sterilization for Reasons of Health in Cases of Otosclerosis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **49**:42 (July) 1939.

12. Guggenheim, L. K.: Therapy of Deafness: Report of Cases, Laryngoscope **53**:441 (July) 1943.

TABLE 1.—*Incidence of Deafness Associated with, or Increased by, Pregnancy in Otosclerotic Women Having One or More Pregnancies, Including Incidence of Familial Deafness*

Patient	Age at Time of Examination	Age at Onset of Symptoms	History of Familial Deafness	Term Pregnancies	Abortions or Other Incomplete Gestations	Deafness Associated with or Increased by Pregnancy	Deafness Not Associated with or Increased by Pregnancy
1. J. J.....	30	29	..	2	..	..	+
2. M. C.....	31	27	..	1	..	+	..
3. R. S.....	25	18	+	1	..	+	..
4. H. S.....	42	32	..	2	..	+	..
5. J. F.....	34	31	..	1	1	..	+
6. A. S.....	52	48	..	1	..	..	+
7. M. D.....	35	28	..	3	..	..	+
8. F. B.....	45	35	+	2	..	..	+
9. M. M.....	37	21	+	1	..	..	+
10. E. M.....	48	23	+	3	..	..	+
11. B. S.....	42	27	..	2	..	..	+
12. M. DiF.....	31	23	+	2	..	..	+
13. W. M.....	44	28	..	2	..	+	..
14. D. D.....	30	28	+	2	..	+	..
15. R. B.....	32	28	..	2	..	..	+
16. E. T.....	32	20	..	2	..	..	+
17. E. A.....	26	22	..	1	..	..	+
18. M. C.....	48	18	+	3	..	..	+
19. J. S.....	44	37	..	1	1	..	+
20. J. L.....	30	25	..	3	..	..	+
21. E. T.....	40	27	..	4	..	..	+
22. B. Y.....	61	31	..	1	..	..	+
23. R. W.....	29	26	+	3	..	..	+
24. S. S.....	59	25	..	1	..	+	..
25. P. O.....	35	30	..	2	..	+	..
26. E. W.....	34	19	..	2	..	..	+
27. A. S.....	32	17	..	2	..	..	+
28. E. K.....	30	23	..	3	..	..	+
29. P. G.....	40	32	..	2	..	..	+
30. M. H.....	38	28	+	2	..	+	..
31. N. H.....	29	25	+	3	..	+	..
32. J. S.....	28	22	+	2	..	..	+
33. S. F.....	45	30	..	3	..	..	+
34. T. G.....	37	25	..	3	..	..	+
35. S. R.....	29	25	..	1	..	+	..
36. Z. D.....	35	34	+	1	..	..	+
37. A. B.....	32	26	..	1	..	..	+
38. F. O.....	52	32	..	1	..	..	+
39. S. M.....	34	26	..	2	..	+	..
40. E. W.....	34	24	+	1	..	+	..
41. D. W.....	41	30	..	1	..	..	+
42. D. S.....	53	33	..	3	..	+	..
43. E. H.....	38	15	..	2	..	..	+
44. V. McM....	33	24	..	2	..	+	..
45. E. H.....	45	18	..	1	..	..	+
46. H. T.....	22	17	..	1	..	..	+
47. B. C.....	28	26	..	1	..	+	..
48. N. C.....	45	33	+	1	..	..	+
49. M. A.....	41	30	..	1	..	..	+
50. I. C.....	28	13	..	1	..	..	+
51. C. D.....	26	16	..	1	..	..	+
52. M. C.....	41	29	..	1	..	..	+
53. M. R.....	27	25	..	1	..	..	+
54. F. Z.....	39	24	..	2	..	..	+
55. Y. J.....	32	9	+	2	..	+	..
56. R. N.....	31	16	..	2	..	+	..
57. F. G.....	35	24	..	1	..	+	..
58. W. B.....	30	20	+	2	..	+	..
59. J. L.....	38	31	..	3	..	+	..
60. W. C.....	40	14	..	3	..	+	..
61. R. S.....	44	14	..	3	..	..	+
62. D. C.....	58	33	..	1	..	..	+
63. R. T.....	57	27	+	3	..	+	..
64. J. B.....	25	24	+	1	..	..	+
65. A. S.....	60	23	..	4	..	+	..
66. L. H.....	31	31	+	1	..	+	..
67. F. D.....	39	?	..	2	..	..	+
68. S. Z.....	44	33	..	3	..	..	+
69. T. L.....	27	22	..	4	..	..	+
70. C. McM....	33	24	+	2	..	+	..
71. S. R.....	42	25	+	2	..	+	..
72. L. W.....	34	34	..	1	..	+	..
73. A. R.....	19	19	..	1	..	+	..

persons will not marry and bear children, or (2) if married, they may avoid having children.

Guild [13] disagreed with those who followed this line of reasoning. He reviewed serially sectioned temporal bones from 1,161 persons by histologic study. Both ears were available from 80.3 per cent. Eighty-one ears (49 patients) showed histologic otosclerosis, but in only 10 of the 81 was there the ankylosis of the stapes necessary for clinical otosclerosis. He concluded that it is useless to try to eliminate the disease by eugenic measures. Further, that all attempts to classify it on the basis of dominant and recessive genes have proved invalid in the face of clinical evidence.

In an attempt to see what information could be added to the situation, a study of case histories was made at the New York Eye and Ear Infirmary.

To insure the validity of the diagnosis of otosclerosis, the survey was restricted to those cases in which fenestration of the labyrinth had been performed or in which after full examination the patient had been judged a suitable candidate for the operation. The review covers the period from 1940 to early 1947. The statistics I shall offer represent married, widowed or divorced otosclerotic women who had had one or more children. The tabulation would have shown higher numbers, but I used only those histories wherein one could accurately determine the number of pregnancies that had existed and what effect, if any, they had had on the patient's hearing.

As table 1 demonstrates, 73 cases were collected. In this table I have presented a brief résumé of data for each patient: the age at the time of examination; the age at the onset of symptoms; the presence or the absence of a history of familial deafness; the number of term pregnancies; the number of incomplete gestations, and, finally, whether the deafness was associated with, or increased by, pregnancy, or was not associated with, or increased by, pregnancy.

The youngest patient was 19 at the time of examination; the oldest, 61. The difficulty in hearing was first noted by 1 patient at the age of 9. This was the earliest onset found. The oldest was 48 when her symptoms appeared. In 1 case the age of onset could not be determined.

The lowest number of term pregnancies was 1; the highest, 4. There were 2 patients who had had incomplete gestations; that is, one each. Neither of these was terminated by surgical interruption.

In table 2 I have summarized for analysis the figures of table 1. The average age of patients at the time of examination was 37. The

13. Guild, S. R.: Histological Otosclerosis, Ann. Otol., Rhin. & Laryng. **53**:246 (June) 1944.

average age of patients at the time deafness was first noted was 25. This figure is in the age range of 16 to 30 years that Nager [2] reported.

The total number of term pregnancies was 134, or 1.8 for each patient. The two abortions that occurred did not cause any remarkable change in the hearing acuity of the 2 mothers involved.

Twenty-eight patients, 37 per cent, gave a history of their deafness being initiated or, if present, increased by pregnancy. A comparison shows that this percentage is higher than that of Nager [2] or that of Shambaugh,[3] but below those of Guggenheim,[4] Allen [9] and Barton.[10]

Forty-five of the patients, 63 per cent, reported that their pregnancies, full term or otherwise, had no effect on their hearing loss. Many of these patients had varying degrees of hearing loss prior to

TABLE 2.—*Analysis of Figures Presented in Table 1*

Total number of patients	73
Average age of patients at time of examination	37 yr.
Average age of patients at time symptoms began	25 yr.
Total number of term pregnancies	134
Average number of term pregnancies per patient	1.8
Total number of abortions or incomplete gestations	2
Number of patients in whom deafness was associated with, or increased by, incomplete gestation	0
Total number of patients in whom deafness was associated with, or increased by, pregnancy	28 (37%)
Total number of patients in whom deafness was not associated with, or increased by, pregnancy	45 (63%)
Total number of patients with familial history of deafness	21 (29%)
Total number of patients with no familial history of deafness	52 (71%)
Cases of deafness associated with pregnancy in which a familial history of deafness was elicited	11 (15%)
Cases of deafness not associated with pregnancy in which a familial history of deafness was elicited	10 (14%)
Cases of deafness associated with pregnancy in which familial history of deafness was not elicited	17 (23%)
Cases of deafness not associated with pregnancy in which familial history of deafness was not elicited	35 (48%)

pregnancy and underwent gestation without any further loss other than that which might be expected from the normal progress of the disease.

Twenty-one women, 29 per cent, gave a history of familial deafness. A cross analysis shows that, of those women who connected their deafness with pregnancy, 11, or 15 per cent, had a history of familial deafness. Against this there were 17 women, or 23 per cent, who connected their hearing loss with pregnancy and had no family history of deafness. Also, there were 10 women, or 14 per cent, who had a family background of deafness but gave birth without ill effect. (This is almost the same figure as that representing women who had a hereditary background and suffered ill effects from birth.) Finally, the women escaping both pregnancy and hereditary background and yet having defective aural reception (35 women, or 48 per cent) formed a group larger than any one of the three groups previously mentioned and larger than any two groups added together; their number indeed was almost equal to the sum of the three.

Since the hereditary effect has no apparent predictable or significant pattern, the conclusion to be drawn from these statistics is that eugenic measures, as suggested by Guggenheim [12] and observed in Europe by Greifenstein,[11] are not the proper solution. The evidence in Fowler's [8] work with identical twins, the conclusions of Barton [10] and Guild [13] and the suggestions to be implied from some of the papers which I have reviewed bear out this trend of thought.

One case, the nature of which the statistics of tables 1 and 2 do not reveal, deserves special attention. The patient (no. 54 of table 1) first noted her loss of aural acuity at the age of 24. It was slowly progressive except for her two periods of pregnancy, during which her hearing improved. After each gestation period was fully passed, her loss of hearing resumed its slow progress until, at the age of 34, she was forced to consult an otologist. This compares with the case that Barton [10] reported. Unfortunately, no audiograms were recorded for the two periods of gestation to prove the point as Brunner [6] was able to do in his third case.

In table 3 I have dissected from table 1 and table 2 those patients in whom the deafness was associated with, or increased by, pregnancy, for separate study. The number and the initials of the patient as they appeared in table 1 are relisted, also the number of pregnancies. With each patient there is presented a brief history of the onset of the hearing trouble and its relation to childbirth.

Scanning the histories, one finds that in many instances the loss of hearing existed prior to pregnancy and was increased by the first or the second pregnancy. Others showed the initiation of their hearing loss with their first, second or third pregnancy. In a few of these the loss was further increased by subsequent gestations.

I have totaled the findings for arithmetic analysis. The result is table 4. In this table there is demonstrated the incidence of deafness in relation to the number of children borne. It has been devised to include those women whose deafness existed prior to childbirth as well as those whose deafness began with it. An attempt has been made to show the effect of further pregnancies once the deafness began to precipitate.

As reported previously, the total number of patients in whom deafness was associated with, or increased by, childbirth was 28. This number can be broken down into four groups according to when symptoms began.

The first group represents those patients in whom symptoms were present prior to pregnancy. There were 11 of these, or 14.7 per cent of the 73 patients studied. In this group of 11, 9 patients had their deafness increased by the first pregnancy; 4 of these 9 suffered additional loss at the second pregnancy. On the other hand, 2 patients from the original 11 did not suffer an increased loss with the first child

TABLE 3.—*Study of Those Otosclerotic Women in Whom Deafness was Associated with, or Increased by, Pregnancy*

Patient	Pregnancies	Comment
2. M. C.	1	Loss of hearing began 3 years prior to childbirth; loss decidedly increased following delivery of first child
3. R. S.	1	Loss of hearing began 5 years prior to childbirth; loss decidedly increased following delivery of first child
4. H. S.	2	Patient noted loss of hearing first while carrying second child; loss decidedly increased following delivery of second child
13. W. M.	2	Loss of hearing first noted after delivery of first child; loss decidedly increased following delivery of second child
14. D. D.	2	Onset of hearing defect noted after birth of second child
24. S. S.	1	Onset of hearing defect noted during latter part of gestation and following delivery of first child
25. P. O.	2	Onset of hearing difficulty followed delivery of first child; deafness increased during and following delivery of second child
30. M. H.	2	Onset of hearing defect followed birth of second child
31. N. H.	3	Onset of hearing defect followed birth of third child
35. S. R.	1	Loss of hearing began 1 year prior to childbirth; loss decidedly increased following delivery of first child
39. S. M.	2	Loss of hearing began 6 years prior to delivery of second child; patient noted no marked change with first child, but birth of second was attended with increased loss
40. E. W.	1	Onset of hearing difficulty began 1 to 2 years prior to childbirth, but was aggravated by birth of first child
42. D. S.	3	Onset of hearing defect noted during latter part of gestation and following birth of third child
44. V. McM.	2	Onset of hearing difficulty began 1 to 2 years prior to childbirth, but was increased by each of 2 pregnancies
47. B. C.	1	Loss of hearing first noted about 1 year prior to childbirth; defect increased following delivery of first child
55. Y. J.	2	Patient first noted slight loss of hearing at age of 9; gradual progression after onset; first child born without any increase being noted, but during gestation and delivery of second the difficulty in hearing increased rapidly
56. R. N.	2	Loss of hearing noted about 9 years prior to childbirth, but difficulty increased markedly during 2 pregnancies
57. F. G.	1	Loss of hearing first noted after birth of first child
58. W. B.	2	Loss of hearing first noted after birth of first child, and hearing loss increased with second pregnancy
59. J. L.	3	Impairment of hearing first noted after birth of third child
60. W. C.	3	Slight loss of hearing since age of 14; slowly progressive; hearing defect increased during first and second pregnancies, but patient had a third without noting any further, or marked, effects as in the first 2.
63. R. T.	3	Slight loss of hearing first noted during late gestation and then more definite loss following birth of third child
65. A. S.	4	Impairment of hearing first noted after birth of second child; hearing defect increased somewhat, but not impressively, by third and fourth pregnancies
66. L. H.	1	Difficulty of hearing first noted during late gestation and after delivery of first child
70. C. McM.	2	Loss of hearing first noted at age of 24; difficulty increased with each of 2 pregnancies.
71. S. R.	2	Loss of hearing first noted after birth of second child
72. L. W.	1	Impairment of hearing first noted after birth of first child
73. A. R.	1	Loss of hearing first noted after birth of first child

but did with the second. Lastly, 1 patient of the 11 was the victim of an increased loss with her first and second children but did not sustain more deafness with her third. This was the only instance in this group of more than two pregnancies.

The second group represents those patients in whom the onset of symptoms was associated with the birth of the first child. There were 8 women in this group, or 10.7 per cent. The 3 women of these 8 who had a second pregnancy encountered further loss in hearing function.

The third group represents those patients in whom the onset of symptoms was associated with the birth of the second child. The mem-

TABLE 4.—*Incidence of Deafness, or Increase in Deafness, in Relation to Number of Children Borne by Otosclerotic Women in Whom Deafness Was Associated with, or Increased by, Pregnancy*

Total number of patients = 78 Number of patients in whom deafness was associated with, or increased by, pregnancy = 28		Deafness Increased by First Pregnancy	Deafness Increased by Second Pregnancy	Deafness Increased by Third Pregnancy	Deafness Increased by Fourth Pregnancy
Number of patients in whom symptoms were present prior to pregnancy	5 (6.8%)	5	..	..	..
	3 (3.9%)	3	3	..	..
	2 (2.6%)	Not increased	2	..	..
	1 (1.3%)	1	1	Not increased	..
	11 (14.7%)				
Number of patients in whom onset of symptoms was associated with first child	5 (6.8%)	..	..	..	..
	3 (3.9%)	..	3	..	..
	8 (10.7%)				
Number of patients in whom onset of symptoms was associated with second child	4 (5.5%)	..	..	..	..
	1 (1.3%)	..	..	1	1
	5 (6.8%)				
Number of patients in whom onset of symptoms was associated with third child	4 (5.4%)	..	..	..	..

bers of this group escaped their first pregnancy without damage. There were 5 of these women, or 6.8 per cent. Only 1 woman of this group had further pregnancies. She had a third and a fourth, reporting some loss of hearing with each but not as evident as with the second.

The fourth and final group represents those patients in whom the onset of symptoms was associated with the birth of the third child. The members of this group escaped their first and second pregnancies without damage. There were 4 women, or 5.4 per cent. None of these had subsequent pregnancies.

Our total for the third and fourth groups, those in whom the deafness began with the second or the third child, was 9 women, or 12.2 per cent. This percentage is below Allen's [9] 29.1 per cent, but compares with Barton's [10] 16.5 per cent.

COMMENT

As I have stated previously, it has been well demonstrated that heredity has little value in predicting the effect of pregnancy on otosclerosis. Also it has been shown that eugenic measures are ineffective in controlling the disease. It is impossible to tell the expectant otosclerotic mother whether or not her child will later have the disease clinically.

That pregnancy and otosclerosis are sometimes linked cannot be denied; most of the studies presented agree, however, that the majority of potentially otosclerotic women undergo pregnancy, or repeated pregnancies, without damage to their hearing.

If difficulty in hearing exists prior to pregnancy, there is the possibility that gestations will increase it. Those in whom this will occur have been shown to be a small segment; the larger portion will escape. Tables 3 and 4 have demonstrated that a woman whose deafness existed prior to pregnancy may have one child without further loss of hearing and then, although this is rare, encounter it with the second. They have also demonstrated that a woman whose deafness existed prior to any pregnancies may experience increased loss with the first and the second child, but have a third without any associated further change in her hearing.

It has been seen that a few women with no deafness prior to pregnancy may have it precipitated by their first child bearing. If these patients have subsequent children, they may or they may not thereby increase their hearing loss. There is no certainty of what will take place.

I have found that some women, with no previous difficulty in hearing, go through their first child-bearing experience unhampered and then have aural symptoms with their second. Others go through two deliveries unscathed and show impaired hearing on delivery of their third child. In both instances the relative number has been small.

The phenomenon is rare, but otosclerosis may improve during pregnancy. I have presented 1 case that seemed to fit this and 2 others from the literature that concur.

For the potentially otosclerotic woman who aspires to a large family, the possibility of impaired hearing is a hazard; but its incidence is unpredictable and uncommon. As pointed out in an editorial in the *New York State Journal of Medicine,*[14] the obvious truth is that little is known about the etiologic factors of otosclerosis, and, in particular, little is known about the relationship of pregnancy to this disease.

14. Otosclerosis in Pregnancy, editorial, New York State J. Med. **45**:2643 (Dec. 15) 1945.

Improvements of recent years, such as socially acceptable hearing aids and the success of the one stage fenestration of the labyrinth, may offer a practical form of relief to those who are afflicted with the more advanced stages of the disease.

In view of the facts summarized in the foregoing pages, and since the disease is not one that endangers life, abortion and sterilization do not appear to be indicated in the average case of otosclerosis complicated with pregnancy.

SUMMARY AND CONCLUSIONS

Reports in the literature since July 1938 that were reviewed show that the effect of pregnancy on otosclerosis and its incidence, as well as the hereditary influences involved, are variable and unpredictable.

Of 73 otosclerotic women studied at the New York Eye and Ear Infirmary, 29 per cent had a history of familial deafness.

The weight of evidence in the literature reviewed and the results of the study presented agree that eugenic measures are ineffective in the prophylaxis of otosclerosis.

Heredity plays little or no part in the effect of pregnancy on otosclerosis.

Hearing loss may be present prior to child bearing and then be further increased by it; 14.7 per cent of the 73 patients studied demonstrated this:

- 6.8 per cent had hearing further damaged by first gestation.
- 3.9 per cent had hearing further damaged by first and second gestation.
- 2.6 per cent had no further damage of hearing with first gestation but did with second.
- 1.3 per cent had further damage with first and second gestation but not with third.

The onset of hearing loss was initiated by pregnancy in 22.9 per cent of the 73 patients studied:

- 10.7 per cent noted onset with first child; 3.9 per cent had a second child, with more loss.
- 6.8 per cent noted onset with second child; 1.3 per cent had a third child with more loss.
- 5.4 per cent noted onset with third child.

In rare instances, otosclerosis improves during pregnancy.

Hearing aids and fenestration of the labyrinth may offer relief to patients afflicted with otosclerosis.

Abortion and sterilization are not indicated in the average case of otosclerosis complicated with pregnancy.

Miss Louise Armao, of the record department, and Miss Eileen McCaffery and Miss Joan Alberque, of the department of audiometry of the New York Eye and Ear Infirmary, gave valuable aid in locating and checking data for this report.

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VOCAL ANALYSIS

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IN TEXTBOOKS of laryngology a chapter on the diagnosis of voice is always missing. The diagnosis of voice is considered the field of singing masters. Only the literature on psychology covers the subject in a more scientific way. However, the fact cannot be denied that the laryngologist sees or, better, hears vocal symptoms every day without being taught how to evaluate them. The visual approach to the appearance and the functions of the vocal cords has become the method of teaching as well as of examining vocal phenomena.

If professional and vocational problems have to be decided in the treatment of singers and speakers, the laryngologist depends on the judgment of a singing master, whose approach again remains a mystery to him and whose scientific qualifications are unknown to him. In addition to this, phoneticians and singers use a different terminology or, better, a different "jargon," so that understanding becomes more and more impossible. The primary need for the task of the analysis of voice is therefore the acoustic approach; the secondary one, an evaluation and a nomenclature which are in accord with the medical way of thinking and which are not exclusively teachable to the musically talented person. Only then will the laryngologist be able to improve on his description of a voice as "hoarse" or "husky."

In human expression the voice plays a singular part. Voice expresses the emotional, articulation the rational, side. The so-called melody of speech is based on voice, which again is the result of respiration and phonation. Since this is so, voice is a vulnerable part in any kind of pathologic condition which disturbs the emotional life. Neurosis and psychosis will be in the scope of laryngologic considerations, as well as artistic problems which have to do with emotional expression. Besides, the vocal function undergoes well known changes due to the conditions of the organs which make up the vocal instrument: the respiratory and phonatory apparatus, as well as the endocrine glands which bring about a change of the larynx. This goes so far that every change in sex life can be diagnosed from vocal changes.

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Instinctively all people listen to vocal phenomena. They know a great speaker or singer from a poor one. They know the symptoms of mutation in boys. They know melancholy when they hear it; they have their likes and dislikes in voices which they hear over the telephone. But who qualifies for voice diagnosis and analysis?

Certainly, a keen ear is necessary. But, besides this, in practice not every good laryngologist can become a good voice diagnostician. All the other medical methods of acoustic examination, e. g., percussion and auscultation, are teachable and learnable. It is some inborn talent and inclination that makes either a surgeon or a psychiatrist. Both have medical talents of equal degree. To become a voice diagnostician, one has to have the faculty of "creative hearing." Among the types of different imagination—the acoustic, the visual, the motoric and the kinesthetic—this faculty is found in mixed acoustic-motorically inclined persons. "Creative hearing" causes an involuntary coordination in the listener's vocal apparatus which corresponds to the speaker's or the singer's coordination. Every one knows the symptom of clearing the throat or even coughing that occurs in an audience which listens to a strained voice. A good voice diagnostician feels, for instance, the sensation of the back of the tongue coming closer to the posterior pharyngeal wall in listening to a "throaty" singing tenor.

This fact of "creative hearing" is entirely unique in medical skill, for in no other diagnostic method does one find any "imitating" reflex reaction in the diagnostician. This subjective sensation belongs to the field of phenomenology, which as a modern conception in psychology is of great medical significance. In hearing a voice one reacts subjectively. One associates, for instance, color images with certain voice sensations. One talks about dark voices, light voices, high voices, black bassos, using synesthesia, a connection between sensations of different senses which is independent of association established by experience.

This sensation is closely connected with the phenomenon of vocal perception and does not substitute for a lacking nomenclature for primary perception. Let it be assumed that one has come to an agreement on color sensation and that except for the color-blind person the terms "red" and "green" have a standardized meaning to everybody. A sound or tone in speaking or singing is, however, a more complex sensation which creates a variety of conditions—sensual ones, physical ones, emotional ones. One reacts with pleasure, displeasure or indifference. This entire complex of psychologic reactions has been called "resonance," and it designates everything that occurs in one who is hearing a voice for the first time. A typical example is the impression which one gets from a voice over the telephone without seeing the speaker. It is in the realm of the psychologist and the psychiatrist. This use

of the term "resonance" is unfortunate and should not be confused with acoustic resonance. Here it will be outlined in the chapter on functional voice disorders.

The voice analysis within the scope of the laryngologist follows the more "old-fashioned" way of the two directions: functional or organic. A specific type of hoarseness leads very often to the diagnosis of an intrathoracic process, e. g., aneurysm, mediastinal tumor or pneumonoconiosis, conditions in which the hoarseness occasionally is the first and only symptom. Laryngoscopy shows the paralyzed vocal cord and roentgen study of the lungs establishes the diagnosis. Here as well as in all organic diseases of the larynx the vocal disturbance might bring the patient to the laryngologist, but the voice as a diagnostic or a therapeutic factor will be of secondary importance after the discovery of the organic condition. This organic disease will be treated, and the voice will be taken into consideration only to measure the value of the treatment. Anyway: the conclusion pursued from hoarseness to intrathoracic pathologic process can be reached by a laryngologist who is not especially trained in the diagnosis of voice.

This becomes different as soon as the hoarseness is caused by an excessively high pitch of voice which is not on an endocrine basis but is, for instance, based on the patient's identifying himself with his mother to whom he has a pathologic fixation. Here one finds again hoarseness, perhaps slightly irritated vocal cords, normal health, normal physique, no disease of endocrine glands. The laryngologist's answer is only too often: "This is functional, this is laryngeal myasthenia, phonasthenia" or any other term which may be supplied in the textbooks to no help for the doctor or for the patient. The laryngologist will have to understand that here he does not face a symptom of disease but a symptom of a fault of personality. This hoarseness does not express a lesion, but the patient expresses himself through the medium of a too high voice.

To use the more recently coined term, "psychosomatic," for these gliding transitions between disease of personality and function cannot be recommended. Voice under any physiologic or pathologic condition remains too much an organ of expression and is therefore an intrinsic part of the body-mind conception in any case. This becomes evident in analyzing the voice of a patient with a psychosis. It is a well known fact that it is from the ruins one learns of the structure and function of each single part. So it is, decidedly, with vocal details.

ACOUSTIC DIMENSIONS OF VOICE

To get a clear idea of what one actually hears, one starts from the fact that speaking and singing are—biologically speaking—identical functions. The only difference is a musical one: that in singing each

syllable has an individual pitch whereas in speaking there is a "glide" through pitches. Since more is known about the structure of the singing voice, one can use the same conceptions and terms for both vocal actions. As outlined at the beginning, voice plays the emotional part in speaking. This "speech melody" is well known to the layman, who is able to differentiate between question and answer only by rising and falling melody even in European languages which he does not understand.

Range.—The range used for speaking is in the lower third of the entire range of an individual voice from the highest tone which the speaker is able to produce down to the lowest. If he uses a higher range for speaking, one more toward the middle third, this means a constant strain on the vocal cords, which are then used under constant constriction to produce this higher pitch. A tired and hoarse voice will be the result. The opposite, a too low range, is a typical symptom in preachers, judges and pediatricians, in short in representatives of paternal, authoritative traits. Such roles are expressed by the "true ring of conviction," phonetically an increased chest register and chest resonance, with the psychologic meaning of father imitation, father identification, which results often in the "I am the law" expression.

In the diagnosis of these phenomena, register and resonance, one finds that instruments and recording devices are of no great help. All one has to use is a trained keen ear.

Register.—The function of registers can be seen with the stroboscope, but only under the unnatural condition which obtains during mirror laryngoscopy. Under "register" is to be understood a physical (acoustic) phenomenon which results from an energetic change in the muscular coordination within the vocal cords. When one sings down from the highest tone which one is able to produce, to the lowest tone—without being a trained singer—one feels and hears a definite break in reaching the middle range and again in reaching the lower range. These breaks or "nodes" indicate a switching mechanism from head register to middle and again from middle to chest register. Anatomically, the head register is produced by isolated vibrations of the inner, the glottal part of the vocal cords, toward their free edges, whereas the lowest tones (the pure chest register) are produced by vibrations of the lateral part. The middle or mixed register which is used in speaking is a well balanced coordination of the whole width of the vocal cords. Metaphorically speaking, if the highest or "head" register is compared to water and the lowest or "chest" register to earth, then there is used in the middle or "mixed" register a mixture of water and earth, a "clay." The more head function or "water" there is in this "clay," the thinner it becomes; the more chest function or "earth," the thicker it will be. In

singing, these different functions are well known and well termed. The thinner mixtures or voices with increased head register are called "lyrical" voices, and the ones with increased chest function "dramatic" ones. Exactly the same takes place in speaking. Voice analysis has to determine range first, but thereafter the analysis of registers gives the best clues.

Figure 1 illustrates the localization of the registers of the vocal cords; figure 2, the relations of the registers.

Fig. 1.—Localization of the registers of the vocal cords: *1*, head register; *2*, chest register; *3*, mixed registers.

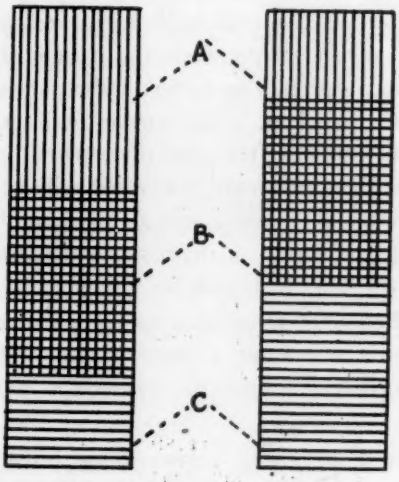

Fig. 2.—Relations of the registers. *A*, head register; *B*, mixed registers; *C*, chest register. The diagram on the left represents increased head register, the "lyrical" voice; that on the right, increased chest register, the "dramatic" voice.

Switching from head to chest register is called "yodeling." Isolated head function is used in "falsetto" and to a large part in "coloratura." Isolated use of chest function is often made in the last syllable of a declarative sentence when one "drops" the voice. Artistically it is used only by certain bassos—for instance, those in the Don Cossack Choir, who produce church bell–like effects with it, whereas in singers of other races pure chest register has only noise character.

As physiology teaches the normal functions of organs, so the analysis of a trained singer's voice reveals a function which surpasses the norm.

It is a complex of coordinations which one may call "ideal physiology." The register blending of artists means the ideal coordination of lateral and medial fibers of the vocal cords. In pathologic disturbance of the singing and speaking voice one observes exactly what is to be expected: namely, that the registers do not blend, that these lateral and marginal functions become isolated. This symptom is called "divergence of registers." It is the most important voice disease in singers and causes innumerable disturbances which only too often are wrongly called "myasthenia."

The old singer becomes unable to mix the two functions. The high range often still sounds normal, the low range is almost flawless, but the described "nodes" become apparent and the mixed or middle range causes difficulties. The same happens when in singing or speaking the wrong range is used. Register coordination becomes almost impossible; the voice gets weaker; its use is a constant strain; anxiety symptoms appear, and the laryngologist is asked for advice. The larynx looks perfectly normal, and the doctor finds four ways out of his dilemma: He uses local therapy for the vocal cords with medication, or he recommends complete voice rest, or he sends the patient to a psychiatrist or to a singing master. Often the latter understands the singer's but seldom the speaker's problem. The former will be able to free the patient from anxiety but not from the divergence of registers. Voice rest is senseless, because afterward the voice will be used in the same wrong way. Medication certainly can do nothing but harm, because of the resulting organ fixation, a neurosis, which misses the necessity of functional change. The main task for the laryngologist is the proper diagnosis. Only then will he be able to recognize the cause of many a secondary laryngitis and many a singer's node. The whole register problem will be considered again in connection with body type and personality.

Resonance.—Chest and head registers are often misinterpreted as chest and head resonance, although register and resonance are entirely different attributes of the voice. Resonance means the transmission of vibrations from one sounding body to another one. In different registers the tone is directed either more toward the chest or more toward the head, where again either the oral cavity or the nasal cavity plays the major part in the transmission. The presence of increased nasal resonance has to be recognized in the evaluation of nasal obstruction and nasopharyngeal processes which might cause "nasality." Haughty voices are nasal ones; hatred is expressed by increased oral and decreased nasal resonance, whereas the expression of love and the desire for closeness is conveyed by increased nasal vibrations, proba-

bly due to slight swelling of the nasal turbinates. It goes without saying that the function of resonance is influenced by any change in the respiratory tract.

Glottal Stroke.—A voice symptom which is easier to diagnose and which causes hoarseness very frequently is the "glottal stroke" or "hard attack," a compression of the vocal cords in starting a tone or a vowel. Froeschels [1] described it as a hyperfunction of vocal cords. The three types of attack have been described frequently as soft, hard or starting with an audible expiration. Here one is less interested in hyperfunction as a symptom than in the cause behind it. The glottal stroke is an almost spastic closure of the glottis immediately before the explosion and vibration in phonation. This overpressure is similar to the increased closing function which one finds in stutterers even while they are not stuttering. Kymograph records of stutterers prove that, for instance, the pronunciation of the letter "P" consists of a closing phase with increased tonus of the lips which is followed by an exaggerated explosion. These phases are not audible, but the visible registration is pathognomonic of stuttering. The glottal stroke is always audible, and the pathologic state behind it is probably the same kind of neurosis which causes stuttering. It has to be regarded as a symptom of displeasure, but besides, as an expression of repression or of aggression. It causes constant irritation of the vocal cords, with hyperemia and swelling, resulting eventually in lasting changes of the free edges. Its counterpart is the soft attack, which, softly pronounced, means pleasure. This gradual approach of the vocal cords without explosion has to be practiced by patients suffering from glottal stroke, but it has to be well understood that these exercises will be senseless without the necessary psychotherapy.

Breathy Voice.—In contrast to this almost spastic compression of the glottis one finds the breathy voice with never completely closed vocal cords, with the symptom of constantly escaping air which is not used to make the vocal cords vibrate. This symptom is called "wild air." Here again hypofunction is not the cause but the result of interesting associations. Semantics, the science of the meaning of words, gives us the explanation. "Inspiration" is common to air and mind. One finds this symptom of wasted exhaled air in "inspired" speakers, in preachers who are so much "inspired" that they expire under pressure as if the amount of exhaled air would indicate the amount of "inspiration."

Respiration.—Certainly, respiration, the power for vocal action, has to be recorded and analyzed to find its influence on pathologic voice.

1. Froeschels, E.: Hygiene of the Voice, Arch. Otolaryng. **38**:122-130 (Aug.) 1943.

The use of belt-pneumographs (one around the chest and the other around the abdomen) will give one an exact picture of any incoordination, which is of great importance especially in functional difficulties of the singing voice. In artistic speaking and singing one faces a unique fact in physiology: The "normal" procedure of breathing described in textbooks is insufficient for ideal vocal activity as trained for artistic purposes. Here one tries to find an ideal balance with regard to thorax-abdomen coordination. The use of tidal air volume in inspiration and expiration is different from the "normal" procedures and figures which are taught by physiologists. To "support" a tone in singing, the diaphragmatic movements are consciously controlled. To do this the abdominal muscles have to be trained to make them controllable. In singers the natural voice is different from the trained one because of the untrained or trained breathing function. The controlled action of the respiratory system "economizes" the action of the vocal cords. These known facts can be investigated by the use of pneumographs.

The traditional separation of breathing types into the female, or chest, and masculine, or abdomen, type cannot be maintained any longer. Through pneumography one learns that in men different body types have different breathing coordination; the asthenic type, for instance, has a typical "female" chest coordination. The analysis of voice has to include the analysis of respiration. The methodologic question of whether the abdominal muscles should be retracted or relaxed in artistic singing during inspiration is still open to discussion. In "bel canto," in the classic Italian technic, the relaxing method, as if one would fill chest and abdomen with air, is the rule.

Rhythm.—Phonation and respiration are rhythmic coordinations. Rhythm and melody are the two pillars which support the whole structure of speech and voice in biologic and psychologic relation. Melody is an exclusive product of voice. Speech melody obeys about the same structural laws which one knows from music. Everything in spoken sentences involves just as much rhythm as one finds in poetry. The only difference is that short and long vowels are less pronounced. One can define rhythm in speech and voice as arrangements of sound phenomena which repeat themselves after about equal periods. The irregularity is important. Like pulse and respiration no living substance produces exact regularity. Exactness is the product of machines and is called time, not rhythm. The main carrier of rhythm in speech is the vowel, not the consonant, therefore the voice. The consonant is the product of articulation, which takes place above the vocal cords. Accents which make a syllable louder, longer and higher are products of the voice. This is the phonetic mechanism of stressing, the effect of increased and longer expiration combined with increased tension and contraction of the vocal cords. In contrast to it: Emphasis affects not one syllable or

one monosyllabic word but a whole sentence, expressing a whole thought. Here instead of one, all the syllables are pronounced higher, louder and longer than those of other sentences. Again one faces an exclusively vocal phenomenon. These rhythmic factors are of great significance in psychologic and psychopathologic considerations of the voice. The pulse influences the voice by cardiopneumatic movements. One finds variations of pressure in the airways with pressure increase during contraction of the heart. The vocal intensity increases and decreases. At the end of expiration one can record a pulsatory tremolo of the voice which becomes audible in vocal fatigue.

Basically the use of rhythm in vocal production starts with the baby's first cry, which already consists of "unrehearsed" rhythmic vibrations. This cry is carried out with a marked glottal stroke, which often has been interpreted as a symptom of displeasure. In reality it is the opening phase of a tightly closed glottis preceding the first inhalation with the intention to remove mucus. The child is preconditioned to rhythm by intrauterine sensations.

Among the "5 R's," respiration, rhythm, resonance, range and register, one finds rhythm the most individual and simultaneously the most sensitive ingredient of the voice. It therefore becomes the best medium for analysis in physiology and pathology.

Pathos.—The physiologic characteristics of rhythmic components can be best analyzed in pathos, which has to be understood here as the emotional kind of speech, not as an affectation. It is the very individual way of exaggerating one's speech attitudes. The movements of pathos are not the small, short, "staccato" ones, but the large, generous ones. The phonetic expression of pathos is complex. Musical components become as pronounced as rhythmic ones. If the melody of the single syllable becomes more pronounced, then speaking is closer to singing. If the rhythmic components stand out, one is more reminded of poetry. In pathos speech is more solemn, more important. The larger movement requires time. Therefore the accents are more marked, and they have something of a "slow motion" character revealing details which would be unnoticed in ordinary speech. In contrast to this pathos are two ways of vocal expression which become familiar to one through the medium of radio and movies.

One hears voices which are overdramatized and voices which are overemotionalized. Both phenomena belong to a pattern of speech which has become typical for these times so that one can call it a "fashion." In both ways of expression one notices hyperfunctions in accents, therefore in pitch, intensity, duration of syllables. Medically speaking, these hyperfunctions cause unnecessary strain of the vocal organs with the effect of vocal irritation in actors and radiospeakers. While true pathos is the expression of dignity and is effected with natural vocal means,

overdramatized and overemotionalized speech is more of an "artificial" coordination which overstresses range beyond the natural means, demanding abnormal balance between expiratory pressure and closure of the glottis.

Speed.—This is a relative conception in speech, again carried mostly by vowels, therefore by voice. A speaker can pronounce a whole sentence by balancing the length of words and of the pauses in between, or he can pronounce single words fast and interpolate long pauses between words. The latter is the typical symptom of dyspnea. But besides the organopathologic evaluation one has to consider speed a highly sensitive psychologic clue, which then in its values has always to be coordinated with measurements from other rhythmic features. As an illustration only, one may take the example of speed of speech in an energetic person. His speed may be identical with that of a highly superficial, unenergetic person. However, by vocal analysis one is able to determine the difference immediately. One only has to measure the individual intensity to find that energy expresses itself with marked dynamic accents whereas superficiality neglects these accents and does not vary the loudness, therefore lacks rhythm.

Melism.—In every conversational speech one gives to one's utterances a certain "touch" by means of special values of pitch, duration, intensity and range, aimed at the listener's reaction. It might be urging, questioning, calming, irritating. This phenomenon is called melism. It is not only individual, but subject to situations, emotions, psychologic influences. It is a signpost to content and direction of speech and voice. In other words, it helps to answer the question: "Why does he say it this way?"; and not only: "Who says it and with what kind of organ?"

According to Buehler,[1a] the product of speech is threefold. It includes expression, appeal and representation. Without changing the sense of the word, the voice alone can express joy as well as grief. One has therefore to consider in the field of voice analysis everything that explains voice as a means of communication. Inclination to communicate and inhibition against it influence the voice in a way which can be recorded and diagnosed.

PATHOLOGIC CORRELATES

From ruins one learns the structure of a building. From voice disorders one learns what voice diagnosis actually means. One learns how to use this long list of diagnostic dimensions for new areas of interpretation. Analysis of a single dimension does not help, but when one learns to hear and to coordinate and subordinate the items, one becomes able to describe the heard voice like a seen picture, because its design

1a. Buehler, K.: Sprachtheorie, Jena, Gustav Fischer. 1934

and color can be termed and expressed. One can now enter areas which otherwise could not be analyzed and interpreted: vocal deviations as signposts of personality deviations, voice syndromes which come about by a complexity of functions.

Endocrinologic Factors and Voice.—The larynx is a secondary sex organ. Every change in sex life can be traced in vocal changes. The mutation in boys is the most drastic one. The lengthening of the vocal cords to about twice their former length results in a pitch which is one whole octave lower than the one before mutation. Hoarseness occurs often before the beginning of an audible change. After the completion of mutation a boy soprano will be a tenor, and a boy contralto will be a baritone or a basso. In girls vocal cords gain about one third of their former length. The drop therefore is only two to three whole steps. Exceptions are the paradoxic mutations in girls who get a range comparable to that of baritones. Every degree in a boy's mutation can be observed, from a drastic, over a mild, to a hardly audible one. If it does not take place at all or only incompletely, one talks about "persistent falsetto voice." This term describes the fact that fully grown vocal cords are not used in their full width; instead vibrations come about only within the medial fibers toward the glottis, producing isolated head register. The other possibility is the organic one; in an undeveloped larynx the vocal cords do not grow in length and therefore keep on producing a boy's range. The first phenomenon is on a purely psychologic basis, the second one on an endocrine one. Then one hears the true eunuchoid voice. Its therapy is not in the field of the laryngologist. Here one is more concerned with the "functional" persistent falsetto voice. The reason has to be found why a person with full-grown vocal cords utilizes only one part of them. Falsetto voice in itself is not pathologic. Every normally speaking man uses it during excitement, when the voice "breaks." Its phonetic opposite is the "true ring of conviction," an increased, almost isolated chest register, which symbolizes balance, quietness. In emotion the breaking voice means loss of mastery over vocal function.

An interesting diagnostic problem is offered by men who imitate female voices, mostly professional singers in variety theaters. Again one sees isolated marginal function of vocal cords with strengthening of the longitudinal extensor muscles through long training. There is no more mixing function possible between the inner edge and the whole width in vibration. In increasing the intensity of a tone the whole pressure is concentrated on the marginal area. The closure of the glottis becomes more and more complete. Therefore the marginal function often produces very strong tones which puzzle the diagnostician, who is uncertain about their nature of being increased head register (falsetto) or really mixed function (from head and chest register).

Some years ago I described the phenomenon of a complete baritone and soprano range in one and the same man, 50 years old, whose two voices were trained professionally. If this man could have built the bridge between his two ranges, he would have had the greatest range ever heard. But the soprano was a well trained marginal function without any gliding transition to the well mixed function of the entire width in the range of a baritone. This man never had a noticeable mutation of voice.

The voice very often heard during drastic mutation has found its way into radio programs to characterize adolescent boys. The too often ridiculed "yodeling" by changing registers within one tone has to be regarded as a difficulty in coordination. The newly acquired body changes require a complete readjustment which is especially difficult during a phase of life which is known as the "awkward age."

Although in girls this phase usually goes on without much dysphonia, menstruation in itself causes vocal trouble which should not be confused with laryngitis. The often noticed inability of an exact intonation and the typical vocal tiredness are not the result of inflammation but of hypotonia. This periodic voice trouble occurs before and during menstruation. During pregnancy similar symptoms occur. Sexual stimulation causes swelling of the nasal mucous membranes as well as of the laryngeal ones, changing the nasal resonance and intonation. The result is a typical "sensual" voice.

In men the beginning, in women the end, of the sex function makes the voice masculine. The normal endocrine influence is revealed more in function than in structure of the larynx. In contrast to this, if insufficient gonadotropic hormones are produced by the pituitary gland, there is underdevelopment of the larynx; the voice is high-pitched and feminine in quality. If androgenic substances are given to female canaries, the canaries sing like males; if given to hens, the hens crow. Testosterone administered for hypogonadism lowers the pitch of voice in sexually immature men. When given to women in the treatment of functional bleeding, it causes occasionally an unwanted low voice.

Thyrolaryngeal syndromes have been reported for years. The symptoms, however, are not characteristic enough to become pathognomonic, at least in cases of milder involvement. In cretinism my observations are in accord with those of Luchsinger.[2] Atrophy of the thyroid gland is accompanied by marked inhibition of the growth of the larynx, together with hypofunction of the sex organs. Typical voice symptoms are: sudden hoarseness, quick tiring and impossibility

2. Luchsinger, R.: Ueber die Stimme und Sprache der Kretinen, Schweiz. med. Wchnschr. **72**:811-817, 1942.

of producing high tones, thus reducing the range. Monotony is another characteristic sign of the voice of the cretin. It is caused by the impairment of hearing, but in addition to this, by the emotional dulness.

The parathyroid glands influence the calcification of the laryngeal cartilages and the calcium deposits within the vocal cords. Since no one knows much about the function of the epiglottis in phonation, laryngologists are still unable to do more than guess at the part of calcification of the petiolus of the epiglottis and its ankylosis in the senile voice. I mentioned in connection with this subject the separation of registers. It depends on the degree of calcification up to what age a singer retains his voice. The horizontal-angular movability of the thyroid cartilage plays an important part.

Dysfunction of the adrenal glands (Addison's disease) almost regularly produces noticeable weakness of the voice. The adrenal cortex influences the muscular tonus of the vocal cords, and the nervous influences on the adrenal glands cause dryness of voice.

The close relationship between endocrine glands and voice brings one back to the more humoral conception of the pre-Virchow period. After all, these voice changes are brought about by secretions of the endocrine glands and not by cell units. New achievements in the field of endocrinology mean new knowledge of the relationship of physique and personality. The vast literature on these subjects has neglected the study of voice almost completely. The reason for this negligence is the technical difficulty of analyzing vocal symptoms. It is because of following the line of least resistance that the more easily recognizable dimensions are described first. Diogenes complained that while people never buy a jug without tapping it to test the sound, when judging men they are content with merely looking at them.

Neurology.—The language and speech of the epileptic person are normal. Unless one hears the speech melody, nothing striking will be noticed. But as soon as one measures the pitch of single syllables in a sentence, the rigidity becomes evident in the melody. In multiple sclerosis the scanning speech is one of the standard symptoms. Often each syllable and each sound is produced separately. However, it is only late in the development of the disease that one's ear is able to register this symptom. Kymographic studies of speech registration reveal it much sooner.[3] In multiple sclerosis the vocal symptoms appear before the speech symptoms. The voice breaks, and pure head register appears. When sound curves are registered on the kymograph, marked deviations are often visible. Progressive bulbar paralysis has characteristic hoarseness and monotony due to incomplete

3. Moses, P. J.: Is Medical Phonetics an Essential Part of Otorhinolaryngology? Arch. Otolaryng. **31**:444-450 (March) 1940.

closure of the arytenoid structures. All these measurable vocal symptoms reveal structural abnormality. Registration of inaudible stiffness of melody in epilepsy has even been used in differentiating epileptic from hysteric seizures.

Psychiatry.—It is in the field of neurosis that voice diagnosis with the help of the described acoustic dimensions brings new areas of interpretation to one's knowledge. Since every emotion influences the voice, since fear, anger, joy are expressed in different pitch, register, resonance, since a child reacts to an overstrong mother fixation by voice imitation, then a drastic influence will leave "scars" which can be traced in vocal symptoms later in life.

A boy who unconsciously identifies himself with his father will do it by lowering his voice in later life as expression of a paternal touch. The more "authority" the father represents, the more pronounced this lowering will be. The simple imitation of voices "with appeal" like those of movie stars or crooners can have a destructive influence. Recently a high school girl aged 16 was seen in the office with dysphonia. She had lost her singing voice completely. She "confessed" that she—a soprano—had tried to imitate her favorite movie actress, who happens to have a low crooner's contralto. An actual neurosis of voice appears when body functions represent conflicts, when a patient escapes to his body, away from problems in life which he is unable to solve. This explains the so-called myasthenia of the larynx which the textbooks describe as a disease in itself, especially in professional speakers and singers. If a school teacher complains of hoarseness which starts soon after the first morning lesson, it is not the mechanical strain, not the wrong speaking technic, but the poor adjustment which causes it. If a personality fails to meet a situation, if no self adjustment can be achieved, it is understandable that the neurotic reaction in professional speakers will hit the organ which is supposed to build the bridge between them and a world to which they cannot adjust themselves.

The influence of emotion on the voice is manifold. The expression of fear for instance is hoarse, high and breathy. The hoarseness is due to an incomplete closure of the glottis. In fear one hesitates to complete a motion. Besides, the dryness of the vocal cords which is based on endocrine changes contributes to the hoarseness. The higher pitch is caused by the contraction of the vocal cords, which can be explained by the instinctive reflex to decrease the body surface. The breathy tone comes about by the change in the inspiration-expiration quotient from pleasure to displeasure, which makes the inspiratory phase longer than the expiratory one.

Personality Structures.—Moreover, the voice expresses moods, traits, drives.[4] The moody cycloid type reveals the up and down phases in a different pitch. His resonance has an increased nasal component. The register is chesty. The speech melody is swinging, gliding through the pitches, not jumping from one to another. The rhythmic qualities are less conspicuous than the melodious ones; therefore his accents are more of a musical than of a dynamic nature. The range of the cycloid voice is wide; the speed is often fast and maintains a melodious variety even in the up phase. In the depressive phase the range is limited, but the so-called monotony does not mean a restriction to one tone, but more a uniform repetition of a small interval.

Vocal signs of the schizoid, the "queer" personalities, are: higher pitch with increased head register, a resonance lacking the intense nasal touch of the cycloid personality, melody with outspoken jumping character, which does not glide through the pitches. The rhythmic features are increased, but the most characteristic symptom is the predilection for unmixed registers. Whole phrases are pronounced in almost pure head register, followed without any transition by others in pure chest register.

To explain these symptoms, the "physiognomics" of voice has to be interpreted. Increased nasal resonance expresses longing for closeness of human and social ties. The desire for distance appears conversely through lack of nasal resonance. Repressions, aggressions, are expressed through the glottal stroke. He who wants to be left alone, who drives for isolation, among the schizoid persons, avoids binding, blending vocal functions. The correlates appear in all the dimensions, like resonance, register and range.

Constitutional Voice Types.—The body types which belong to these voice types are best classified, according to Sheldon,[5] as follows:

Endomorphs—predominance of soft roundness—with the vocal signs of the cycloid personality.

Mesomorphs—predominance of muscle, bone, connective tissue—with the vocal signs of the schizoid personality.

Ectomorphs—predominance of linearity and fragility—with the vocal signs of the schizoid personality.

The cycloid and schizoid persons are normal persons with personality traits like "prestages" of the psychotic condition of schizophrenic and manic-depressive patients. Sane or insane—the constitutional types are

4. Moses, P. J.: The Study of Personality from Records of the Voice, J. Consult. Psychol. **6**:257-261, 1942.

5. Sheldon, W. H.: The Varieties of Human Physique, New York, Harper & Brothers, 1940, pp. 37-46.

the same; therefore a certain pattern of vocal dimensions exists in the prestage and is "enlarged" under psychotic conditions.

It is most interesting to compare these voice types with traditional representatives of personality types on the opera stage. Here one differentiates first, according to registers, the lyrical and dramatic singers as head or chest functions are increased. Second, one differentiates, according to range, tenor, baritone, basso, soprano, mezzosoprano, contralto. The hero sings tenor, the villain baritone or basso. The baritone and the basso often represent paternal figures. The woman of intrigue sings contralto. The tradition goes on for several hundreds of years. The phenomenologic reaction of the listener to this typologic classification must have a natural rather than an artificial basis. The singing voice can be considered as the speaking voice "under a magnifying glass." The resonance in the listener is about as predicted by the evidence which the analyst's ear and recording and measuring devices give him.

SUMMARY

A group of acoustic dimensions of the voice is explained which have physiologic and pathologic correlates. The laryngologist's ear can be trained to perceive and conceive the "5 R's": register, range, resonance, rhythm, respiration, as well as pathos and melism. The so-called functional voice disturbances like myasthenia of the larynx and dysphonia can be explained and analyzed with these dimensions, because each one expresses certain personality traits. Therefore they are helpful in showing the relationship between voice type and constitutional type.

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INVASION OF ETHMOID SINUSES BY ADENOMA OF THE PITUITARY GLAND

Report of a Case

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SINCE invasion of the ethmoid sinuses by tumors of the pituitary gland is a relatively infrequent occurrence, it seems wise to record the following case in the literature.

REPORT OF CASE

M. B., a 56 year old white woman complaining of a sinus infection, was first examined by me on April 29, 1947. She had had three operations on the left side of the nose and, after the last, extensive cauterization with silver nitrate.

On examination, widespread adhesions were observed between the left side of the septum and the inferior middle turbinate. The middle meatus and the posterior portion of the nose could not be clearly visualized, but there was purulent discharge coming from above the adhesions. Roentgenograms showed sclerosis in the ethmoidal region and clouding of the left antrum, the left ethmoidal region and the sphenoid sinuses.

On May 4, the patient came into the accident room after a hematemesis of 1,500 cc. of bright red blood. She was admitted to the hospital on the medical service and treated with supportive therapy. During the next few days, gastroscopic and roentgenoscopic studies were carried out. No explanation for the bleeding was found.

On May 26 she was again admitted to the hospital, and the following day, under local anesthesia, the adhesions in the nose were severed. Considerable tissue was removed from the anterior end of the left middle meatus. This tissue was friable; it did not have the appearance of nasal polyps, and it bled profusely. Bleeding was controlled by absorbable gelatin sponge ("gelfoam"). Pathologic examination of the removed tissue showed that it was a chromophobe adenoma of the pituitary gland. Roentgenograms indicated a much enlarged sella, with erosion at its base and with fused clinoid processes. Thorough investigations revealed no neurologic abnormalities and normal visual fields. The patient soon felt perfectly well.

Roentgen therapy was begun one week later. The region of the anterior fossa and the paranasal sinuses was irradiated through three portals, one anterior, one left and one right lateral, each of which measured 6 by 8 cm. The patient's eyes were protected with lead. The treatment continued for about three weeks,

during which each field received a total of 1,200 r in six 200 r doses measured in air. The factors used were 200 peak kilovolts, 20 milliamperes, 50 cm. skin target distance and Thoraeus filter equivalent to 2 mm. of copper and 1 mm. of aluminum.[1]

SUMMARY

A 56 year old white woman has been found to have invasion of the ethmoid sinuses by chromophobe adenoma of the pituitary gland, with no demonstrable neurologic lesions. An explanation for her gastric bleeding was not found. At the time of writing she is asymtomatic.

The Johns Hopkins Hospital.

1. Dr. George Farber, of the Johns Hopkins Hospital, administered the roentgen therapy.

ROLE OF THE SEPTUM IN RHINOPLASTY

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THE PURPOSE of this paper is to discuss the interrelationship of septal and external nasal malformations and the rationale of rhinoplastic reconstruction. Although an extensive literature is available on descriptive and topographic nasal anatomy, contributed by such authorities as Zuckerkandl,[1] Zarnicko [2] and others, comparatively little that is authentic can be found on the surgical relationship of the malformed septum and the deformed external nose. Even Joseph's valuable book on rhinoplasty is not an exception in this regard.[3]

During the last few years, as rhinoplasty became widely popular and rhinologists displayed great interest in this field, new views and methods appeared in the literature. In a number of articles the reconstruction of the septum, particularly, became the chosen target of discussion, the role played by the septum as a supporting structure receiving special attention.[4] Some challenged the accepted view that the position of the cartilaginous dorsum and tip depends on the septum. It was also claimed that saddling of the dorsum following a high submucous resection is not the result of lack of septal support but is due to scar contracture. The fact that the alar dome maintains its forward position after the excision of the anterior part of the septal cartilage is adduced as proof that the cartilaginous septum does not provide any support for the tip.

On the basis of this hypothesis, procedures of septal reconstruction have been presented in which the entire septal cartilage is removed and, after being straightened, is reinserted between the mucoperichondrial flaps, supposedly to prevent subsequent contracture and saddling.

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1. Zuckerkandl, E.: Anatomie normale et pathologique des fosses nasales et de leurs annexes pneumatiques, Paris, Masson & Cie, 1895.

2. Zarnicko, C.: Die Krankheiten der Nase, und ihrer Nebenhöhlen des Nasenrachenraumes mit besonderer Berücksichtigung der rhinologischen Propaedeutik für praktische Aerzte und Studirende, Berlin, S. Karger, 1910.

3. Joseph, J.: Nasenplastik und sonstige Gesichtsplastik: Nebst einem Anhang über Mammaplastik und einige weitere Operationen aus den Gebiete der äusseren Körperplastik, Leipzig, Curt Kabitzsch, 1931.

4. Fomon, S.; Syracuse, V. R.; Bolotow, N., and Pullen, M.: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141-156 (Aug.) 1946.

A procedure in which the bent anterior part of the septal cartilage is excised and reinserted as a free graft into the columella to provide support for the tip is also often advocated.

Some of these theories and methods of reconstruction are unwarranted, as they have no basis either in clinical observation or in surgical anatomy.

The plastic surgeon usually sees a septal deflection associated with external deformity. The relationship between them being close, their repair should be planned as a single project to be carried out in one or more surgical stages according to the situation. To treat the deformity of the septum as a purely rhinologic problem independent of the external

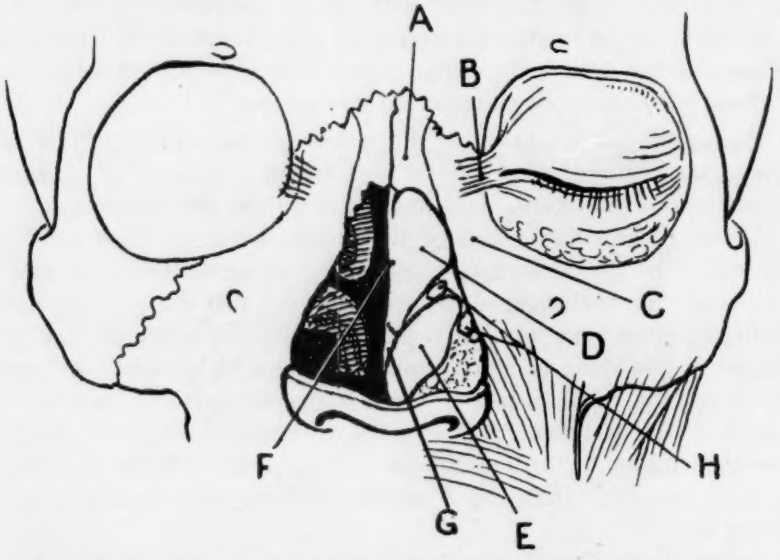

Fig. 1.—Surgical topography of the nasal cartilages and the frontal processes of the maxillas. *A* indicates the left nasal bone; *B*, the left palpebral ligament; *C*, the frontal process of the left maxilla. *D*, the left upper lateral cartilage (triangular cartilage). *E*, the left lower lateral cartilage (alar cartilage). *F*, the septal cartilage. *G*, the exposed, most vulnerable part of the dorsum of the cartilaginous septum. *H*, the minor alar cartilages of the left side.

deformity is contrary to the fundamental principles established through extensive experience in corrective rhinoplasty.

It is advisable to review some of the anatomicophysiologic facts concerning the septum that are involved in pathologic distortions. A detailed description of septal structures and their function has been presented elsewhere.[5] Here only the principal characteristics are con-

5. Maliniac, J. W.: Rhinoplasty and Restoration of Facial Contour with Special Reference to Trauma, Philadelphia, F. A. Davis Company, 1947.

sidered, mostly those of the cartilaginous framework, knowledge of which facilitates understanding of the mechanism and the treatment of these deformities.

THE CARTILAGINOUS FRAMEWORK

The fibrocartilaginous structure of the nose is based on the position and the interrelationship of the cartilages, on the one hand, and the bony framework, on the other (fig. 1). It is essential to consider the following cartilages enclosed in the piriform aperture: (1) the septal cartilage, (2) the triangular cartilages and (3) the alar or quadrangular cartilages.

Septal Cartilage.—The topography of the inferior border of the septal cartilage has important anatomic significance in that it normally

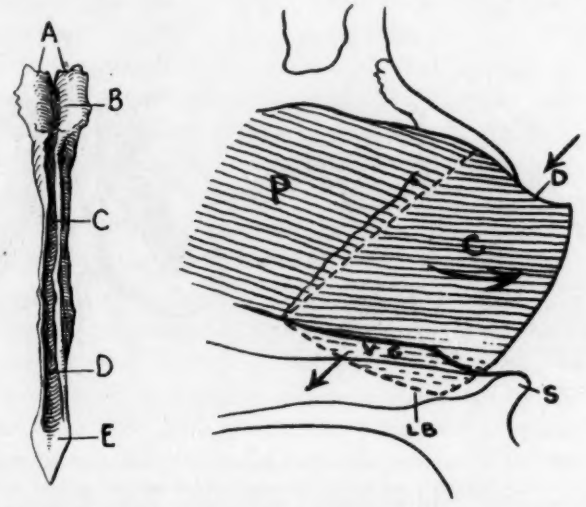

Fig. 2.—Left: Vomerine groove as seen from above. *A* indicates the alae of the vomer; *B*, the vomer–sphenoid bone articulation; *C*, the region in which the vomer articulates with the perpendicular plate of the ethmoid bone; *D*, the groove for the septal cartilage; *E*, the ungrooved surface of the maxillas and the nasal spine.

Right: Septum dislocated from the vomerine groove, with arrows showing the direction of the trauma. *C* indicates the twisted septal cartilage; *D*, the depression at the dorsum; *L B*, the lower border of the septum, out of the vomerine groove (*V G*); *S*, the nasal spine; *P*, the perpendicular plate.

rests in the groove along the anterior border of the vomer (fig. 2, *left*). Since the maxillary bone and the nasal spine in front of the vomer are flat and slightly oblique, they afford a sliding surface for the septal cartilage. The perichondrium and the periosteum form a fibrous capsule, which accounts for the mobility of the septum in this area. Thus the latter, well affixed in the bony posterior angle and along the vomerine groove, becomes loose over the maxillary bone and the anterior part of the nasal spine. The tendency of the anterior part of the septum

to become dislocated at the slightest trauma is due to this laxness. The lower segment of the alar or quadrangular cartilage also represents the zone of active growth, and at this level occur most of the spurs which extend along the vomerocartilaginous articulation (fig. 2, *right*).

Triangular (or Upper Lateral) Cartilages.—In the region of the nasal tip the mobility of the septum is partially impeded by the triangular cartilages attached to the bony aperture. This attachment serves as a support for the suspension of the septum as well as for that of the nasal tip and contributes to the elevated position of the alar dome in the absence of the anterior part of the septum.

The triangular cartilages, each fused on one side to the dorsal border of the septum, are attached on the other side to the nasal bones and

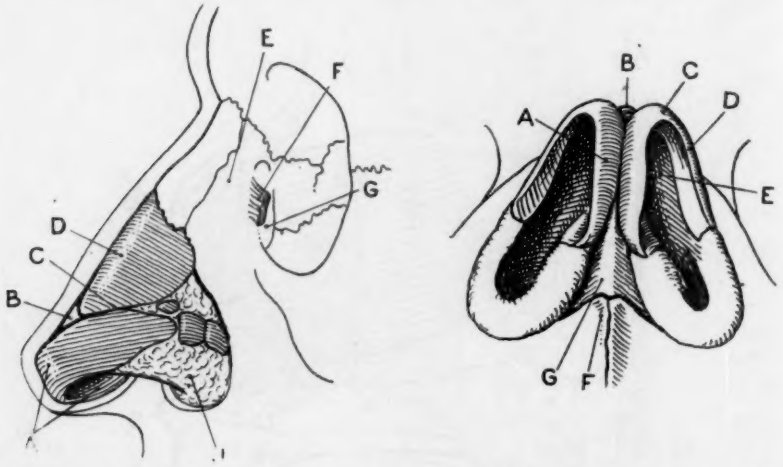

Fig. 3.—Left: The surgical interrelationship of the lateral cartilages and the septum. The alar cartilages (*A*) are suspended by an unyielding skin covering and by loose connective tissue extending to the septum and the triangular cartilages. At *B* the septum is directly beneath the skin and most vulnerable to injury. The main incision in rhinoplastic procedures is made through the intercartilaginous space (*C*). The upper lateral cartilage (*D*) is fused to the septum at its dorsal border, thus reenforcing it. Corrective infracturing of the frontal process (*E*) is done anterior to the palpebral ligament (*F*), which protects the lacrimal sac at *G*. *J* indicates the noncartilaginous area of the nostril.

Right: The close interrelationship of the septal and the tip cartilage seen from below. *A* indicates the medial crus of the alar cartilage; *B*, the exposed portion of the dorsum of the septal cartilage; *C*, the dome of the alar cartilage on which the form of the nasal tip depends; *D*, the lateral crus of the alar cartilage; *E*, the vestibule. *F*, the nasal spine on which the base of the septal cartilage (*G*) rests; the latter supports the columella.

frontal processes by means of fibrous tissue originating in the perichondrium and the periosteum.

Intimately connected with the septum on one side and with the piriform aperture on the other side, these lateral cartilages provide not only the suspension of the cartilaginous dorsum but also protection

against trauma. However, in the rhomboid area above the alar dome the septal dorsum is exposed between the divergent ends of the triangular cartilages (fig. 3, *left*). This constitutes the "weak spot" of the dorsum, which is located directly above the alar dome. It is at this place that the septal dorsum usually sinks in when septal cartilage is removed in toto or excessively in a submucous resection. This depression occurs immediately after surgical intervention and therefore is not due to scar contracture.

Alar (or Lower Lateral) Cartilages.—These cartilages participate in the structure of the nasal tip—an anatomic entity—to a great extent by forming the dome. But the shape and the position of the nasal tip also depend on the configuration of the anterior part of the septum along the columella and the rhomboid dorsal segment (fig. 3, *right*). The inner branches of the alar cartilages, which form the columella, are only loosely connected with the anterior nasal spine; so they do not account for the forward position of the nasal tip. This is determined by the anterior part of the septum, supported on the maxillas and protruding forward, carrying the alar cartilages, winglike, on the two sides The anterior part of the septum is attached to the alar cartilages by loose connective tissue and the membranous septum. Thus the alar dome is suspended by the upper lateral cartilages from above and supported by the lower lateral cartilages from below.

In reconstruction of the tip, the mutual adherence of the vestibular lining and the alar cartilages is easily demonstrated during their dissection. In the absence of the anterior part of the septal cartilage the skin covering the tip, a covering which is continued by the vestibular and mucous membrane lining, forms an additional suspension support for the alar dome. Retraction of the columella and depression of the rhomboid segment of the dorsum, which will determine the shape of the nasal tip, never fail to take place when the anterior part of the septum is missing.

A CLASSIFICATION OF SEPTAL DEFORMITIES

In general, there are three main types of septal deformity associated with external malformation of the nose: (1) septal deflection with oversized hump deformity, (2) crushed septal fracture with depressed external deformity and (3) septal deflection with external lateral deviation.

TIMING OF SEPTAL CORRECTIONS

When septal deflection is associated with external deformity, the order of the corrections may come into question. As a general rule, if the deflection is such as to interfere with the successful performance

of corrective rhinoplasty, it should be repaired simultaneously with, or prior to, the latter. In old depressed fracture of the septum, for example, the fragments usually override; the result is a thickening of the septum. In this case, thinning and resetting of the latter and provision of support for the nasal tip can be done at the same time at which a graft is inserted in the depressed area of the dorsum, or they may be done previously. A high septal deviation which would interfere with narrowing of the dorsum should be corrected simultaneously with the external deformity. On the other hand, in oversized deformities with lateral displacement, in which a pronounced deflection of the bony septum is accompanied by a large bony hump, the submucous resection should follow the rhinoplasty. Otherwise, the freely mobilized

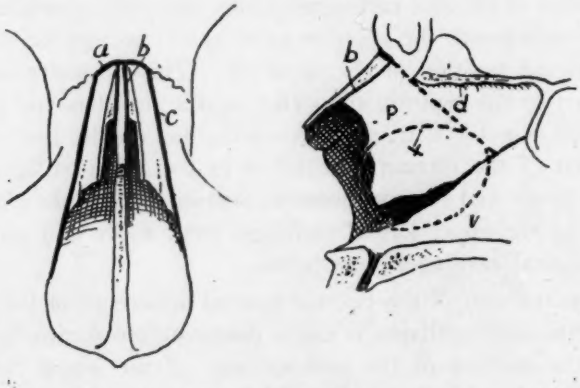

Fig. 4.—A condition in which an obstructing bony septum should be removed after rhinoplasty: a deflected bony septum associated with an oversized bony bridge that requires narrowing. Here the bony septal support should be straightened, if possible, and not excised. If excision is required, it should be done after corrective rhinoplasty and consolidation of the refractured bony bridge. At the left are shown the lines of the infracturing of the bony bridge following removal of the latter's oversized part: (*a*) along the frontal juncture, (*b*) between the nasal bones; (*c*) along the frontal processes.

At the right is shown an area of excision (*x*) which endangers the bony bridge (*b*), disconnected as this is from frontal and lateral attachments. The perpendicular plate (*p*) sinks along the line *d*. The vomer is indicated by *v*.

bony bridge, detached from the frontal suture line and the frontal processes and deprived of bony septal support, may sink in (fig. 4). I have observed this condition in a number of patients. To afford partial relief from obstruction, the cartilaginous septum can be reset in the midline during the corrective procedure. A high cartilaginous deflection can be straightened by crisscrossing and partial excision of cartilage in the same operative stage.

REPAIR OF SEPTAL DEFLECTION IN AN OVERSIZED TYPE OF DEFORMITY

From the rhinoplastic point of view it is primarily important to preserve sufficient support for the newly reconstructed dorsum and nasal tip. Wide septal resection is not usually required to reestablish the patency of the nasal passages. Often this can be effected by centralization and thinning out of the septum, although deflected thickened parts may be removed in zones of absolute safety distant from the dorsum and the nasal tip. As the dorsum and the columella are approached, the safety factor is diminished, the resection of cartilage becoming increasingly dangerous (fig. 5). To prevent accidental excessive removal, no pulling instruments should be used during the process of resection. Only a knife, angular scissors or a cutting septal gouge should be employed for this purpose.

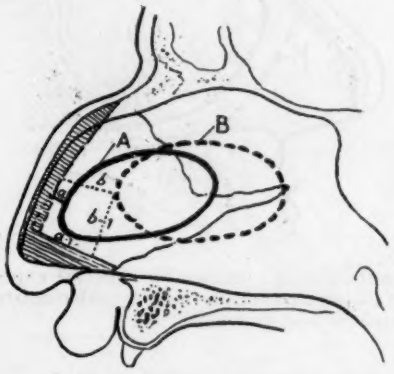

Fig. 5.—Safety zones in submucous resection with relation to external deformity. *A* indicates an area of resection endangering the stability of the cartilaginous dorsum; *B*, a safe area of resection. At *a* an inadequate cartilaginous bridge will be left by excision *A*. At *b* a wide cartilaginous support will be left by excision *B*, insuring safety of the dorsum. The shaded area represents the segment to be removed in oversized deformity. The area of safe septal excision must be based on the dimensions of the normal nose, even in the presence of oversized deformity not corrected at the time of submucous resection. The proportions *a-1* and *a-2* apply to the shortening of the septum.

My usual procedure is to expose the cartilage widely through a long incision extending anteriorly from the dorsum to the floor of the nose and anteroposteriorly along the deflected part (fig. 6). In deflections above the floor of the nose a horizontal incison is made along the top of the spur, and the mucous membrane is separated from above downward.

When shortening is required at the same time, the anterior incision is made about 2 to 2.5 cm. (¾ to 1 inch) from the anterior septal border. To prevent bone or cartilage from being exposed in the nasal cavity, the horizontal incision is made low enough so that the mucous

membrane can be shifted from the floor of the nose to cover the line of infraction.

The part to be resected posteriorly is best exposed by means of a long-blade speculum.

Bent septal cartilage below the dorsum is left attached to the muco-perichondrium unilaterally, and the deviation is corrected by making

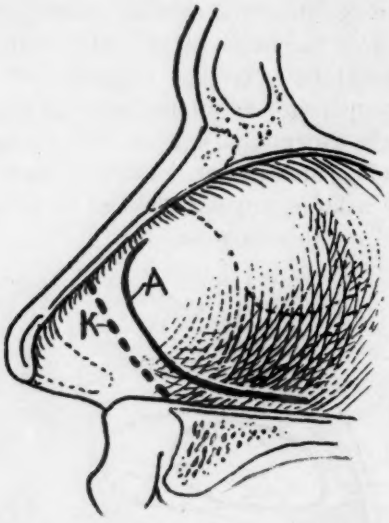

Fig. 6.—*A* indicates the author's incision for adequate exposure of the septum; *K*, the "classical" Killian incision commonly used by rhinologists. The latter does not allow for sufficient exposure and drainage.

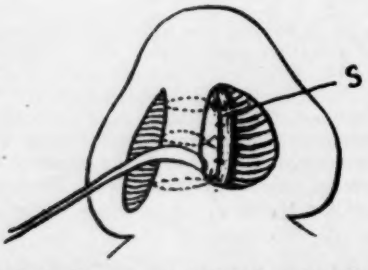

Fig. 7.—This drawing shows an isolated columellar strut (*S*) inserted through an anterior vestibular incision. Such a strut must be inserted deep in the columellar pocket and must rest firmly on the maxillary bone. Mattress sutures passed through the columella and the graft (*S*) solidly affix the latter, preventing formation of dead space.

crisscross incisions through the adherent cartilage. This brings the septum to the center without the risk of excessive removal of cartilage. The procedure is of particular value in children, in whom undue sacrifice of septum leads to faulty development.

REPAIR OF CRUSHED SEPTAL FRACTURES IN DEPRESSED EXTERNAL DEFORMITY

In this condition the separation of mucoperichondrial flaps is often laborious because of fibrous adhesions and lack of cleavage. It is advisable to leave some fibrocartilage attached to the flap on each side in order to prevent perforation of the flap. Generally in this circumstance the anterior part of the septal cartilage is displaced or missing. After enough of the fibrocartilage has been removed to supply an adequate strut to support the tip, it is shaped and inserted through a columellar or a vestibular incision, a few mattress sutures maintaining

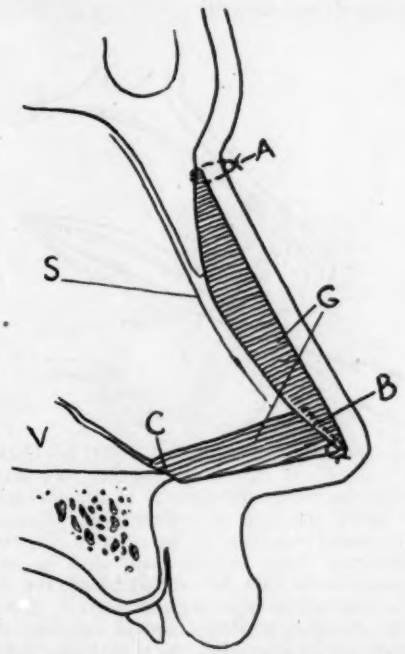

Fig. 8.—Provision for septal support in crushed deformities. The forms and the relationships of the dorsal and columellar grafts (*G*). The sides of the dorsal graft continue the lateral bony walls; the upper end (*A*) is immobilized and affixed with an external suture. It is necessary that there be wide contact in a sagittal direction between the two grafts at *B*, where they are sutured. The lower end of the columellar strut (*C*) must rest firmly on the nasal spine. *S* indicates the septum; *V*, the vomer.

it in place (fig. 7). In most cases, however, the septal fibrocartilage removed is insufficient to furnish a buttress for the tip, and a free graft is required. This graft must be properly immobilized to insure its survival. This is accomplished by placing mattress sutures behind the graft and applying pressure dressings on both sides of the septum. Simultaneously correction of the depression of the dorsum can be carried out (fig. 8).

After trauma to the nose of a child one must look for crushed deformity of the septum. In childhood, displacement of the cartilage occurs more commonly than fracture and thickening. If support for the tip and cartilaginous bridge is required, it should be provided early so that normal development of the nose may be assured. The reparative procedure is similar to that previously described.

Since in childhood the nose grows continuously, immediate permanent correction is frequently impossible, even if an autogenous graft is inserted. As yet no proof has been forthcoming that such a graft continues to grow adequately within the child's nose. An isogenous septal graft may be used temporarily.

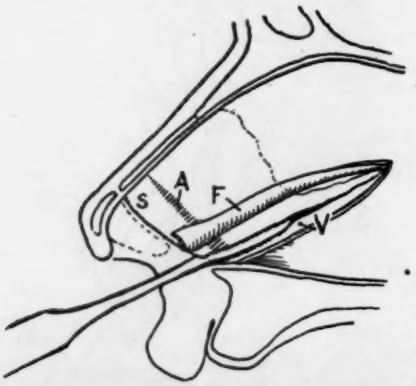

Fig. 9.—Resetting in the presence of limited septal angulation and displacement. A mucoperichondrial flap (*F*) is raised by angular incision so as to give wide exposure of the septum; the horizontal branch of the incision is made low so as to permit the flap to cover the line of infracturing. Excision along the lower border permits the subluxated cartilage to be reset in the vomerine groove (*V*). If necessary, the dislocated bony septum can also be refractured and reset mesially. A cartilaginous strip (*A*) is excised along the angle of deflection to effect resetting of the septum in the sagittal plane. Throughout, the septum remains attached to the mucoperichondrium on the opposite side, assuring stability of the reset parts, which are held in place with mattress sutures.

REPAIR OF LATERAL DEVIATION OF THE SEPTUM WITH EXTERNAL LATERAL DISPLACEMENT

In nasal fractures with lateral displacement, dislocation of the septum is almost invariably present. One may observe slight anterior deflection, and/or subluxation, and/or unilateral and bilateral deviations with or without torsion of the dorsum.

Displacement of the Anterior Part of the Septum.—In the presence of limited angulation and displacement, an incision made along the angle of deflection, with or without excision of a vertical segment, will suffice for complete straightening of the septum. In case of lateral traction,

the mucous membrane along the floor of the nose is excised (fig. 9). Posterior submucous resection is done if necessary.

Pronounced Angulation of the Septum with Torsion of the Dorsum.—Often the methods advocated for the repair of this condition are inadequate. Statements are frequently made to the effect that complete correction of a twisted dorsum is impossible and should never be attempted. My experience is that a simultaneous correction of the twisted type of septal and dorsal deformity should always result in a complete straightening of the curvature. In Metzenbaum's [6] procedure, an anterior displacement of the septum is released with straightening of the nasal tip, but a deflected cartilaginous dorsum cannot be reset. The

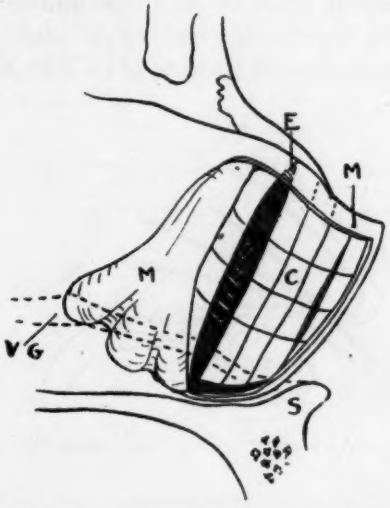

Fig. 10.—Procedure for the repair of pronounced deflection of the dorsum of the cartilaginous septum. A mucoperichondrial flap (*M*) is incised along the dorsum, the columella and the floor, permitting wide exposure of the twisted cartilage (*C*). The latter is freely mobilized after an excision (*E*) has been made along the angle of deflection extending from the dorsum to the nasal floor. Crisscrossing of the cartilage is done along the entire height. The septum is reset in the vomerine groove (*V G*) after being resected along its lower border to permit free mobilization. In pronounced obstructions there may be resection of the posterior part of the septum. The mobilized septum remains attached to the mucoperichondrial flap on the opposite side. All parts are maintained in place with mattress sutures throughout the entire height of the septum and anteriorly along the columella. *S* indicates the nasal spine.

same statement applies to procedures in which reliance is placed on excision of the deflected cartilage and insertion of a free columellar graft.

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6. Metzenbaum, M.: Replacement of the Lower End of the Dislocated Septal Cartilage Versus Submucous Resection of the Dislocated End of the Septal Cartilage, Arch. Otolaryng. **9**:282-295 (March) 1929.

The exact procedure which I employ depends on the extent of the septal deflection and dorsal twist.[7] The bridge in the sagittal plane cannot be completely straightened until all secondary bends are released either by vertical excisions of narrow segments along its entire height or by crisscrossing when curvatures are less marked. All these manipulations are done without separation of the mucoperichondrial flap on one side, which serves for splinting the reshaped septal cartilage. The traction of the shortened mucous membrane on the concave side is eliminated by an incision made along the floor of the nose. The resulting raw surface may be large enough to require a thin skin graft to prevent recurrence of traction. If shortening of the septum is required, it is carried out at this time.

The flaps are held in place by mattress sutures. To reenforce the immobilization of the septum, a wired septal splint may be employed, which dispenses with endonasal packing (figs. 2 [*right*], 7, 10 and 11).

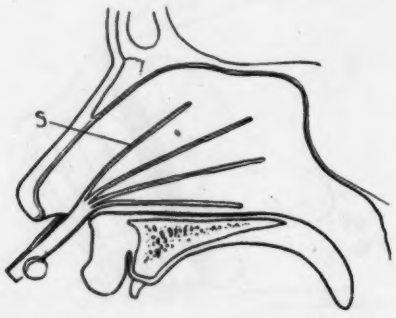

Fig. 11.—Wire splint (*S*) in place along the septum.

SUMMARY

The anatomic and surgical relationships between septal and external nasal deformities are discussed.

The position of the nasal tip is determined by the manner in which the alar dome is suspended from above and the septum supported from below. Additional suspension of the tip is provided by the nasal skin covering and lining.

If septal support is missing, the remaining suspension will maintain the alar dome in position, but the columella and the lower part of the cartilaginous dorsum will retract.

In the correction of oversized deformities the extent of submucous resection should be determined by safety zones that allow for sufficient support of the dorsum.

7. Maliniac, J.: Fracture-Dislocations of the Cartilaginous Nose: Anatomicopathologic Considerations and Treatment, Arch. Otolaryng. **42**:131-137 (Aug.) 1945.

In the repair of crushed deformities the missing columellar strut should be replaced, if possible, with an autogenous septal graft.

In the correction of lateral deviations the septal cartilage should never be freely excised but should remain attached to the mucoperichondrial flap on one side.

In the timing of repair, if a deflection interferes with successful performance of rhinoplasty, it should, as a rule, be corrected simultaneously with, or prior to, the latter.

The treatment of a pronounced vomerine deflection associated with a marked oversized bony deformity should follow corrective rhinoplasty to prevent a possible sinking of the dorsum.

11 East Sixty-Eighth Street.

OPHTHALMIC CONDITIONS REFERABLE TO DISEASES OF THE PARANASAL SINUSES

HENRY M. GOODYEAR, M.D.
CINCINNATI

FROM a rhinologic point of view I wish to mention first the following observations made in cases of retrobulbar neuritis. When apparently secondary to rhinitis, the central blindness, unilateral, is often first observed by the patient in the morning on awakening; hypothetically, it follows an inflammatory change which has occurred in the optic nerve during the night ,while the patient was in a recumbent position.

On rhinologic examination, and even on surgical opening, the ethmoid and sphenoid sinuses appear to be normal. In these cases in which examination yields apparently negative results, vision frequently returns one or two days after the opening of the sphenoid sinus and the posterior ethmoid cells on the side of the optic lesion, even though no gross pathologic change was evident.

However, in the past fifteen years I [1] have observed that the injection of 27 per cent iodized oil ("iodochlorol" [peanut oil with iodine and chlorine added]) into the sphenoid sinus will produce the same spectacular result as is obtained by surgical intervention. I have repeatedly seen marked clearing of vision within a period of twenty-four to forty-eight hours after the injection of this oil, and this is now routine treatment in such cases. Should chronic infection be found on injecting the oil into the antrums, these are dealt with surgically.

I have seen total blindness follow surgical opening of the sphenoid sinus and the posterior ethmoid cells, and I wish to present a specimen which I found while working over the heads of some cadavers (fig. 1). The optic nerve crosses freely through a posterior ethmoid cell, suspended in a cylinder of thin bone; a heavy cord could be passed about the nerve without difficulty.

Dr. B. T. Horton, of the Mayo Clinic, has been a strong advocate of the use of a solution of histamine phosphate (2.75 mg. in 250 cc. of isotonic sodium chloride solution U. S. P.), which is injected intra-

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Read at the Twenty-Eighth Annual Meeting of the Virginia Society of Ophthalmology and Otolaryngology at Virginia Beach, Va., May 10, 1947.

1. Goodyear, H. M.: Diagnosis and Treatment of Nasal Sinus Conditions in Relation to the Eye, West Virginia M. J. **39**:270 (Aug.) 1943.

venously at 30 drops per minute once or twice daily in the treatment of retrobulbar neuritis. On the other hand, he has mentioned the possible danger of the use of typhoid vaccine, in that it may fix the retrobulbar neuritis, preventing any further recovery.

Headaches referable to disease of the nasal sinuses are usually worse on one side or are unilateral but are not apt to be confused with histamine-induced headaches, which are always distinctly unilateral, with attacks occurring most frequently after midnight, causing the patient to arise with severe pain, accompanied typically by profuse lacrimation on the affected side.

Fig. 1.—Specimen showing suspension of the optic nerve as it passes through a large posterior ethmoid cell.

I have seen tularemia mistaken for an infection [2] of the sphenoid sinus with deep pain about the eye. The calico-like appearance of the cornea plus some swelling of the glands of the neck aroused my suspicion and led to the information that the patient had been exposed to rabbits. An agglutination test was positive for tularemia.

I should like to mention a lesion which often comes to the attention of the ophthalmologist, primarily because of the acute swelling and edema about the eye following an acute infection of the frontal sinus. This lesion is probably most frequently seen in young persons who have been swimming a day or two previous to the onset. It is always

2. Goodyear, H. M.: Tularemia Simulating Symptoms of an Acute Ethmoid, Sphenoid Infection, Laryngoscope **50**:1019 (Oct.) 1940.

dangerous, and Courville [3] stated that subdural empyema secondary to an acute primary infection of the frontal sinuses is four times as frequent as subdural infection following acute exacerbation of a chronic infection of the frontal sinuses. The progress of the condition is rapid,

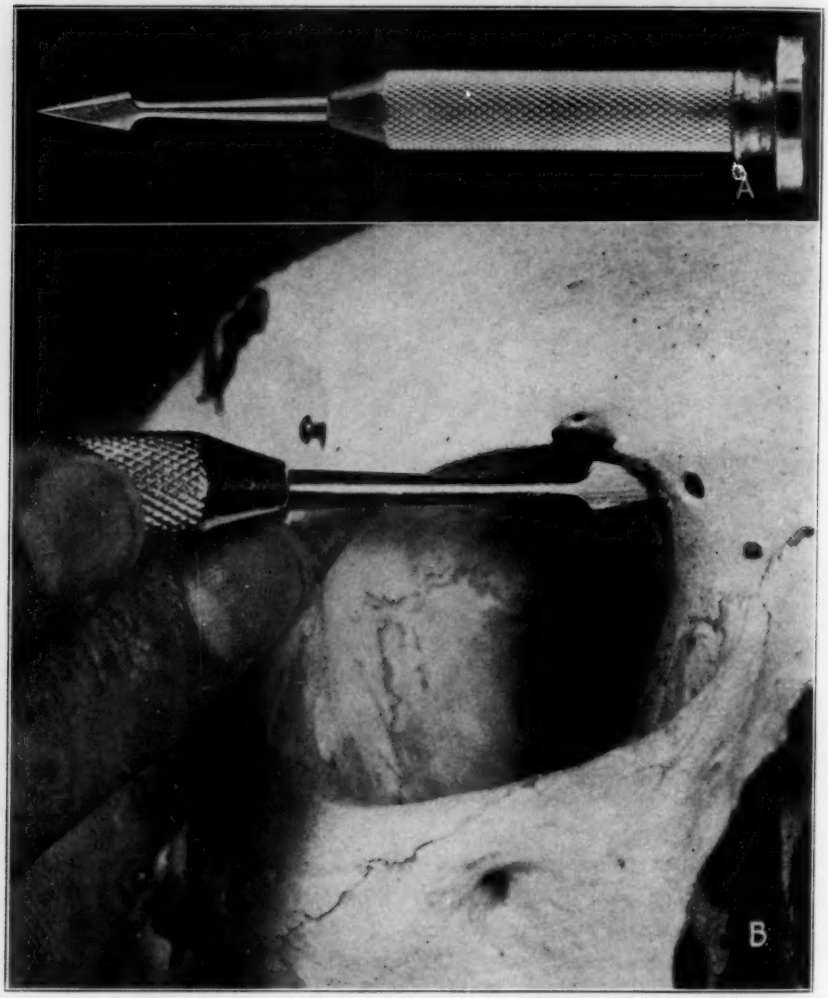

Fig. 2.—*A*, type of nail drill recommended for an emergency opening of the frontal sinus. (This instrument is furnished by Max Wocher & Company.)
B, perforator, directed almost at right angles to the sagittal plane, entering just posterior to the medial part of the orbital ridge.

3. Courville, C. B.: Subdural Empyema Secondary to Purulent Frontal Sinusitis: Clinicopathologic Study of Forty-Two Cases Verified at Autopsy, Arch. Otolaryng. **39**:211 (March) 1944.

and not infrequently death occurs with meningitis within three or four days after the onset.

Chemotherapy plus external drainage is indicated for the severe forms, and I should like to suggest a simple way of relieving the pressure in the frontal sinus. A sharp nail perforator (fig. 2*A*), such as that used to relieve accumulation of blood under a finger nail, is directed almost at right angles to the sagittal plane and a small hole is made just posterior to the medial orbital ridge (fig. 2*B*). A small curved incision is made primarily just below the eyebrow, *without elevation* of the periosteum. This is important, as any unnecessary elevation of the periosteum will produce periostitis and undue scarring on healing.

Through the opening made, there is a welling of pus or serous fluid; the sinus can be irrigated with isotonic sodium chloride solution U. S. P. or penicillin solution and a small catgut drain inserted for forty-eight hours. This is an important procedure, giving great relief and comfort, and should be performed before an intraorbital abscess or meningeal symptoms occur.

Recurring corneal ulcers demand a careful survey of the paranasal antrums, and such a survey does not mean that examination of a roentgenogram will suffice. Iodized oil should be injected into each antrum and a nose-chin and a lateral (slightly oblique) view made. Even a thickening of the antral membrane of 2 mm. is significant,[4] and, other foci of infection, such as those of teeth and tonsils, having been eliminated, a large antral window should be made under the inferior turbinate bone and the antrum packed with 5 per cent iodoform gauze (½ inch strip, which is to remain from four to eight days.

I have seen many ophthalmic lesions which had been permitted to progress for months because roentgenologic reports had indicated that the sinuses were normal. These same sinuses, when iodized oil was injected, showed definite lesions on roentgenograms. One of my patients, a young man almost totally blind, had spent months in some of the best clinics in this country without being relieved of bilateral retrobulbar neuritis. Yet, when "iodochlorol" was injected into the antrums, infection was found in one antrum. This was treated surgically and an improvement of vision followed within forty-eight hours. Six months later the man was able to drive his car, and he has remained well.

One must remember that the antrum is the most important of all the paranasal sinuses, and infection of it is always accompanied by some infection of the ethmoid cells. The latter recover in a large percentage of cases with proper care of the antrum.

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4. Semenov, H.: The Surgical Pathology of Nasal Sinusitis, J. A. M. A. 111:2289 (Dec. 10) 1938.

I regret that small doses of insulin, 3 to 5 units (hypodermically) daily or twice per week, are not used more frequently in the treatment of lesions of the eyes, especially corneal ulcers. Insulin stimulates metabolism, body chemistry and healing.

I wish to emphasize that surgical drainage of an intraorbital abscess is not usually indicated before there is evidence of fixation of the movement of the eye. Drainage then should be provided by means of a curved incision extending from just below the eyebrow downward, midway between the inner canthus of the eye and the midline of the nose. The incision is carried through the periosteum, and no attempt is made to enter the sinuses. Chemotherapy is started early. Should suppuration continue, sinusotomy can be performed after the acute infection subsides. Most patients, however, recover without further surgical treatment or recurrence. In a few cases in which the middle meatus was sufficiently wide, I have drained an intraorbital abscess intranasally by biting away the ethmoid cells and removing the lamina papyracea.

Acute suppurative ethmoiditis occurring in an infant is frequently mistaken for dacryocystitis. If the primary incision is made high and a subperisoteal abscess is found in the region of the ethmoid cells, the lacrimal sac need not be explored. Simple incision and drainage will usually suffice, together with chemotherapy, without the nasal sinuses being disturbed.

I have seen severe hematoma arise in the orbit after injury had been done to the anterior ethmoidal artery while an ethmoidectomy was being performed. Hematoma may follow a fracture extending into the orbit. One should not hesitate too long in doing external aspiration or incision and drainage before the severe stretching of the optic nerve by the hematoma leads to permanent blindness.

I [5] have ligated the anterior ethmoidal artery successfully by an intraorbital incision in intractable recurring epistaxis with blood coming from an area high in the nose.

Patients with tumors in the paranasal sinuses often go to the ophthalmologist first because of disturbed vision and pain of the eye, and, frequently, double vision. If the tumor is in the antrum or the ethmoid cells, it must be dealt with radically by means of electrocoagulation and radium. One should not hesitate to sacrifice the eye in furtherance of a sufficiently radical procedure. Often the severe pain produced in the eye by a malignant tumor of the antrum makes enucleation of that organ a humane procedure.

An osteoma producing swelling in the orbit and double vision is easily identified by roentgenogram and must be removed by a combined

5. Goodyear, H. M.: Nasal Hemorrhage: Ligation of the Anterior Ethmoid Artery; Case Report, Laryngoscope **47**:97 (Feb.) 1937.

radical external frontal and ethmoid sinusotomy. Once an osteoma has reached the stage at which ocular symptoms are evident, chronic suppuration of the frontal sinus has occurred, and I [6] have advocated the use of Ingals' gold tube, to be inserted at the time of operation before the wound is closed, to remain for the life of the patient, insuring a patent nasofrontal opening.

Mucoceles of the ethmoid and frontal sinuses represent one of the lesions with which every ophthalmologist should be familiar lest he

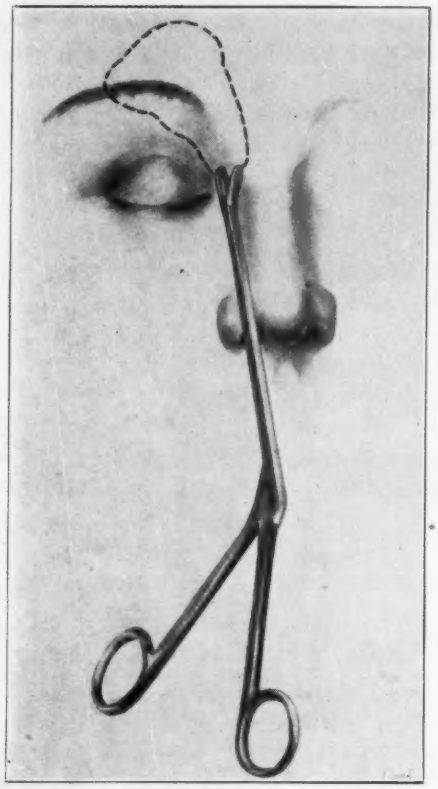

Fig. 3.—General outline of a mucocele of the frontal sinus with its lower part extending into the anterior ethmoid area, where the base may be grasped with an instrument and removed.

subject the patient, or permit the patient to be subjected to, an external operation which is disfiguring, usually unnecessary, and often the beginning of prolonged episodes of sinus disease.

The patient usually comes to the ophthalmologist with double vision and a swelling at the inner angle of the orbit; occasionally, the forehead is

6. Goodyear, H. M.: Surgery of the Frontal Sinus, Laryngoscope **57**:340 (May) 1947.

bulging and the eye is displaced downward and outward. Paper shell crepitus may be elicited on pressure. Roentgenograms show a faint outline of a tumor, not dense as in a case of osteoma, and absorption of bone may be evident.

The treatment consists in removing the lower end of the mucocele (fig. 3), which has usually extended downward into the anterior ethmoid area, with *minimum disturbance* of the epithelial lining of the mucocele.[7] The tumor externally is pressed back into normal position by means of the finger, and the double vision soon disappears. The patient usually leaves the hospital in one or two days.

It may be necessary to do a resection of the nasal septum and to remove the lateral half of the middle turbinate bone, but one should do as little surgical work as possible in treating this particular lesion. Nature already has lined the intranasal extension with epithelium far better than one could ever hope to do by skin grafting.

If double vision should persist after an external frontal sinusotomy, the wound should be reopened under local anesthesia, and, with the patient focusing on a finger, the superior oblique muscle should be reattached (slightly overcorrected) at a point where the double vision ceases. I have had this experience in 2 cases with immediate and excellent results.

556 Doctors' Building.

7. Goodyear, H. M.: Mucocele in Frontal and Ethmoidal Sinuses: Simplified Surgical Treatment, Ann. Otol., Rhin. & Laryng. **53**:242 (June) 1944.

TUBERCULOSIS OF EAR, NOSE AND THROAT TREATED WITH STREPTOMYCIN

A Report of Twelve Cases

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AND
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AT THE present writing there have been reported 3 cases in which tuberculous involvement of the larynx and the hypopharynx was treated with streptomycin. There have been no reports of tuberculosis of the ear or the nose treated with this antibiotic agent.

Figi, Hinshaw and Feldman [1] reported 1 case in which tuberculous laryngitis was treated with streptomycin, with apparent healing. Hinshaw, Feldman and Pfuetze [2] have reported 2 additional cases in which tuberculous involvement of the hypopharynx and the larynx was treated with streptomycin, with apparent healing.

This paper is a report of 12 cases of tuberculosis of the ear, the nose or the throat treated with streptomycin at Fitzsimons General Hospital, Denver. The patients include 6 with laryngitis, 1 with pharyngitis and laryngitis, 3 with otitis media, 1 with bilateral mastoiditis and 1 with involvement of the nasal septum.

TUBERCULOSIS OF THE LARYNX

In this group there are 6 cases—4 with apparent healing and 2 with improvement of symptoms and visible lesions.

CASE 1.—E. J. F., a 36 year old master sergeant, was seen for the first time in the eye, ear, nose and throat clinic on Nov. 14, 1946. He complained of soreness of the right side of the throat, present for the past two weeks, and of persistent hoarseness of several months' duration and stated that he had noted intermittent hoarseness for the past five years.

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From the Eye, Ear, Nose and Throat Section, Fitzsimons General Hospital, Denver.

1. Figi, F. A.; Hinshaw, H. C., and Feldman, W. H.: Treatment of Tuberculosis of the Larynx with Streptomycin: Report of a Case, Proc. Staff Meet., Mayo Clin. **21**:127 (March 20) 1946.

2. Hinshaw, H. C.; Feldman, W. H., and Pfuetz, K. H.: Streptomycin in Treatment of Clinical Tuberculosis, Am. Rev. Tuberc. **54**:191 (Sept.) 1946; Treatment of Tuberculosis with Streptomycin, J. A. M. A. **132**:778 (Nov. 30) 1945.

The larynx could not be visualized properly by indirect means. Direct laryngoscopy was performed on November 21. The finding was a "sessile mass involving approximately the middle two thirds of the right vocal cord, covered with superficial ulcers." The rest of the larynx was apparently normal. A specimen was taken from the mass for biopsy. Stained sections were made, and a report of tuberculous laryngitis was returned. Administration of streptomycin was started on December 6 (0.5 Gm. being given daily in divided doses by nebulizer).

Laryngoscopy was performed on December 6; the finding was similar to that of November 14, with the addition of slight edema and superficial ulceration of the middle half of the left true vocal cord. Three days after the administration of streptomycin was started, the soreness of the throat had diminished, and eleven days later it had disappeared. On December 23 laryngoscopy revealed "slight proliferation of granulation tissue along the edges of both true cords." No ulceration was noted. On Feb. 4, 1947, laryngoscopy revealed slight injection of the blood vessels of both true cords. The same finding was noted on February 18. On February 25 the cords were reported as being "negative to inspection." Since that time examinations have revealed no pathologic conditions.

A roentgenogram of the chest, taken July 19, 1946, revealed "infiltration of the upper lobes of both lungs, with a 2 cm. cavity underlying the second rib anteriorly on the left." On August 6 left anterior pneumothorax was produced. As late as Jan. 24, 1947 no change was noted on the right side. On the left there was 60 per cent pneumothorax, with a cavity, 1 cm. in size, still present. Weekly smears and cultures of concentrated sputum have shown tubercle bacilli to the present time. The patient is still being treated with streptomycin.

The final diagnosis is tuberculous laryngitis, apparently healed.

CASE 2.—J. S., a 39 year old soldier, was seen in the eye, ear, nose and throat clinic for the first time on May 27, 1946. He complained of hoarseness, present since February 1946. Indirect laryngoscopic examination revealed a diffusely injected, edematous lining of the left true cord, extending into the left ventricle. No paralysis of the cords was present. The impression at that time was chronic laryngitis, believed to be tuberculous because of definite pulmonary lesions, sputum containing tubercle bacilli and laryngeal findings. The patient was given streptomycin (1.8 Gm. intramuscularly daily) from July 10 to November 8. At that time he was symptom free, and the larynx was normal in appearance.

The initial roentgenogram of the chest revealed infiltration of both lungs with considerable cavitation of the upper lobe of the right lung and minimal bronchogenic spread involving the upper lobe of the left lung. Administration of streptomycin was started July 10. Roentgenograms of the chest taken one and two months after the institution of therapy revealed no essential change. Roentgenograms taken three months later showed a decrease in the size of the lesion in the upper lobe of the left lung. No change was noted in the involvement of the right lung.

Weekly smears and cultures of concentrated sputum were positive for acid-fast bacilli and tubercle bacilli, respectively, throughout the entire course of therapy except for one reported "negative."

The final diagnosis was chronic laryngitis, probably tuberculous, apparently healed.

CASE 3.—O. L., a 27 year old soldier, was seen in the eye, ear, nose and throat clinic for the first time on Sept. 28, 1945. He had noted intermittent hoarseness since April 1945, unassociated with pain. At that time examination of the ears, the nose and the throat revealed essentially normal conditions except in the larynx.

Laryngeal examination with mirror disclosed prominent thickening, slight injection and grayish discoloration of the entire laryngeal mucosa. The true cords were thickened and were covered with superficial ulcers on the apposing surfaces. In the anterior commissure there was a small granulomatous lesion with subglottic extension. This patient was not seen again in the eye, ear, nose and throat clinic until June 6, 1946. At that time the laryngeal findings were essentially the same as previously noted.

The patient was treated with streptomycin from June 18 to October 18 (1.8 Gm. being given intramuscularly daily). Two weeks after streptomycin therapy was begun (July 2) the larynx was essentially normal and remained normal on subsequent examinations.

The initial roentgenogram of the chest revealed dense consolidation in the upper lobe of the right lung with marked atelectasis and a large cavity, also minimal involvement of the left cardiophrenic region. As stated, administration of streptomycin was started June 18. Roentgenograms taken one month later revealed no essential change, and those taken two months later showed a slight decrease in the volume of atelectasis of the upper lobe of the right lung and a slight but definite decrease in the size of the cavity. Those taken three months later revealed no change.

Smears of concentrated sputum showed acid-fast bacilli until June 30 and none after that date. Cultures of concentrated sputum regularly revealed tubercle bacilli until July 2; thereafter they occasionally did until the eighty-fifth day of therapy; then they became consistently negative.

Hearing was normal as tested with whispered and spoken voice and on audiometric examinations during the entire time of observation.

The final diagnosis was chronic tuberculous laryngitis, apparently healed.

CASE 4.—A. A. was seen for the first time in the eye, ear, nose and throat clinic on Dec. 11, 1946. He complained of hoarseness present since November, unassociated with pain. Examination of the ears, the nose and the throat showed essentially normal conditions except in the larynx. Mirror examination of the larynx revealed a moderate amount of injection of the blood vessels of both the true and the false cords, bilaterally, and small superficial ulcers along the medial borders of both true cords. The patient was given streptomycin (2 Gm. intramuscularly daily), starting Jan. 3, 1947, and is receiving streptomycin at the time of writing.

Laryngeal examination on February 21 revealed no abnormality of the larynx.

The initial roentgenogram of the chest revealed pneumoperitoneum, with both sides of the diaphragm elevated to the level of the ninth intercostal space, a cavity with a fluid level in the right intraclavicular area, 2.5 cm. in diameter, and a 1 cm. cavity in a dense area of infiltration overlying the left hilus at the level of the seventh posterior rib.

The patient was given streptomycin, starting on Jan. 3, 1947. One month later roentgenograms revealed a definite decrease in the size of the cavity in the left midlung region, a decrease in the size of the cavity in the upper lobe of the right lung and also a decrease in the extent and the density of the exudative lesion in the entire right lung and the left midlung area. Three months later roentgenograms revealed a further decrease in the size of both the aforementioned cavities.

Weekly smears and cultures of concentrated sputum have been positive to the time of writing.

The final diagnosis is tuberculous laryngitis, apparently healed.

CASE 5.—A. B. was seen for the first time in the eye, ear, nose and throat clinic on Oct. 8, 1946. He complained of hoarseness, present for the past several months,

and severe pain on swallowing. Examination of the ears, the throat and the nose gave essentially negative results except for marked involvement of the larynx; the arytenoid structures were strikingly enlarged, especially the right, blocking off the right piriform fossa. The membrane of the arytenoid cartilages appeared to be smooth, but its blood vessels appeared diffusely injected. The right false cord was enlarged to an impressive degree; the surface was irregular and grayish. The right true cord could not be seen except for its most posterior portion near the vocal process. This area was moth eaten in appearance and grayish. There was fixation of the right true and false cords. The epiglottis was moderately enlarged, with a definite cobblestone appearance due to superficial ulcerations. On November 20 a specimen taken from the right false cord for microscopic examination revealed tubercles and acid-fast bacilli. In the pathology laboratory the diagnosis was chronic tuberculous laryngitis. This patient was treated with streptomycin from November 3 to December 11 (1 Gm. per day by nebulization). The aerosol treatment was discontinued at the patient's request because of pharyngeal irritation. Aerosol treatment was reinstituted Jan. 27, 1947 and continued until March 6. Intramuscular injections of streptomycin (2 Gm. daily) have been given since March 6.

Laryngeal examination on Jan. 24, 1947 showed no essential change. Examination on March 13 revealed enlargement of the right ventricle. No ulceration was present. The epiglottis appeared to have irregular edges. There was no other sign of pathologic change. Both vocal cords were normal in appearance. There was no history of pain or hoarseness at this time.

The initial roentgenogram revealed scattered linear and nodular infiltration involving the right lung from the apex to the seventh rib and the left lung from the apex to the fourth rib. Streptomycin treatment was started for the first time on Nov. 3, 1946. Repeated roentgen examinations revealed no essential change through Feb. 11, 1947.

Monthly smears and cultures of concentrated sputum have shown acid-fast bacilli and tubercle bacilli to the time of writing.

The final diagnosis is chronic tuberculous laryngitis and tuberculosis of the epiglottis. As to the results of therapy—the condition is improved, not healed, to date.

CASE 6.—E. S. R., a 32 year old soldier, was seen for the first time at the eye, ear, nose and throat clinic on Dec. 30, 1946. His complaint was that of hoarseness and pain in the throat, beginning about November 1. These symptoms had been progressively aggravated until the time of the first examination.

The otolaryngologic examination showed essentially normal conditions except for the following findings: The epiglottis was definitely enlarged and moderately diffusely injected, with a striking cobblestone appearance along the entire border. Both vocal cords were covered with a whitish slough. The movements were normal. The patient has been treated with streptomycin to the time of writing (1 Gm. in divided doses being given by nebulization daily). On Jan. 27, 1947 he was symptom free except for slight hoarseness. Indirect laryngoscopic examination revealed moderate enlargement of the epiglottis with the cobblestone appearance previously noted. There was a piling up of granulation tissue in the interarytenoid area. Both the true and the false vocal cords were covered with a whitish slough. The movements were normal. On April 2 indirect laryngoscopic examination revealed a definite decrease in the size of the epiglottis. A slight cobblestone appearance was present still. There was no

granulation tissue in the interarytenoid area. Both the true and the false cords, however, were moderately enlarged, the linings of both being smooth and the vessels injected.

The initial roentgenogram of the chest, taken Dec. 23, 1946, revealed a diffuse nodular and linear infiltration of the parenchyma of all lobes of both lungs and an area of rarefaction in the region of the first anterior intercostal space on the right and the second anterior intercostal space on the left. The administration of streptomycin was started on Jan. 4, 1947. Subsequent roentgenograms of the chest revealed essentially the same findings as those described.

Weekly smears and cultures of concentrated sputum showed acid-fast bacilli and tubercle bacilli until Feb. 13, 1947, at which time they became "negative."

The final diagnosis is tuberculosis of the larynx and the epiglottis. As to the result—the condition is improved, not healed, to date.

TUBERCULOSIS OF THE PHARYNX AND THE LARNYX

There is 1 case, with apparent healing.

CASE 7.—R. R., an officer, was seen for the first time in the eye, ear, nose and throat clinic on Feb. 13, 1946, with a history of difficulty of swallowing and sore throat since December 1945.

The examination of the ears, the nose and the throat gave essentially negative results except for a slight diffuse injection of the blood vessels of the pharynx and a pinhead-sized grayish elevated area in the right posterolateral aspect of the pharynx. This small area of tissue was removed with punch forceps. The report of the pathology laboratory was "tuberculous pharyngitis." Within the next four months the process spread, involving the tonsillar fossae, the uvula, the epiglottis and the arytenoid structures. The vocal cords could not be visualized. During May 1946 a 1 per cent procaine hydrochloride solution and alcohol were injected into the left superior laryngeal nerve, giving only temporary relief. Pharyngeal sprays of a 10 per cent cocaine hydrochloride solution were used also for pain, giving only temporary relief.

Streptomycin was given from June 18 to Oct. 18, 1946 (1.8 Gm. intramuscularly daily and 0.5 Gm. daily by nebulization). Three days after the start of streptomycin therapy there was a remarkable diminution in the dysphagia and the soreness of throat. Four days later there had been an amazing improvement in the local pharyngeal condition. Only one small area of visible pathologic change remained, at the base of the left side of the uvula. Fourteen days after the beginning of therapy the pharynx appeared to be entirely normal except for a slightly grayish tint to the left tonsillar fossa. There was no visible evidence of past disease. One hundred and twenty-five days after the administration of streptomycin was started, there was no longer any evidence of infection. Laryngoscopic examination gave negative results. The patient was symptom free—as he had been two weeks after the start of therapy.

The initial roentgenogram of the chest revealed infiltration of the upper lobes of both lungs. The upper lobe of the right lung was partially collapsed by pneumothorax. A few small cavities were present in the upper lobe of the left lung.

Administration of streptomycin was started on June 18, 1946. One month later there was complete disappearance of pneumothorax. The lesion of the upper lobe of the right lung was more discrete and slightly decreased in size. Two months later there was noted a further decrease in the lesions in the upper lobes of both lungs.

Weekly smears of concentrated sputum were negative for acid-fast bacilli after five weeks of therapy. Weekly cultures of gastric content were negative after the second week. Weekly cultures of concentrated sputum were negative except for the first and eleventh weeks of therapy.

Prior to the time at which streptomycin began to be used in this case (intramuscularly and by nebulization) the patient complained of severe and persistent sore throat and dysphagia. These symptoms did not improve except temporarily after frequent sprayings with cocaine hydrochloride solution or even after injections of procaine hydrochloride solution and of alcohol. However, after three days of streptomycin therapy the patient already was noticing improvement, and after two weeks he had complete relief. Subsequent to that time the symptoms did not recur during the entire time the patient was under observation.

The final diagnosis was tuberculous pharyngitis, apparently healed.

TUBERCULOSIS OF THE NASAL SEPTUM

There is 1 case in which tuberculosis of the nasal septum improved after administration of streptomycin.

CASE 8.—V. W., a 21 year old member of the Women's Army Corps, was seen for the first time at the eye, ear, nose and throat clinic on Dec. 12, 1946. At that time a minute perforation, approximately ⅛ inch (3 mm.) in diameter, was seen in the anteroinferior portion of the nasal cartilaginous septum. By Jan. 28, 1947 the perforation was 1 cm. in diameter and revealed small irregular grayish ulcers, partially covered with blood crusts. A biopsy of tissue from the border of the perforation was reported by the pathology laboratory as demonstrating tubercles and acid-fast bacilli.

The application of streptomycin packs (0.5 Gm. daily) was started on February 14, and the patient continued to receive streptomycin treatments until March 7. At that time she was transferred to another hospital.

On March 4 examination revealed the perforation to be of the same size. However, the border of the perforation was smooth and normal in color.

The initial roentgenogram of the chest revealed an area of parenchymal infiltration in the upper lobe of the right lung at the periphery of the first anterior intercostal space. The final roentgenogram revealed no change. Smears and cultures of concentrated sputum and cultures of gastric content, taken at weekly intervals were negative for tubercle bacilli throughout the patient's stay in the hospital.

The final diagnosis was tuberculosis of the nasal septum, improved.

TUBERCULOSIS OF THE MIDDLE EARS

There are 3 cases—2 with apparent healing and 1 with no improvement.

CASE 9.—C. H. H., a 32 year old top sergeant, was seen for the first time in the eye, ear, nose and throat clinic on Sept. 3, 1946, with pain in the right ear following a third stage thoracoplasty (with removal of ribs 7 and 8) done on Aug. 23, 1946 and spontaneous purulent drainage of the right ear, starting Sept. 1, 1946.

The examination of the ears, the nose and the throat disclosed essentially normal conditions except that in the right ear there was a thin serous discharge coming from a small central perforation of the drum. Routine culture of the

discharge revealed anaerobic diphtheroid bacilli. The patient was treated with penicillin, without improvement. On September 17 examination revealed that the right tympanic membrane was distinctly thickened and was bulging posteriorly. Two small, dry, central perforations could be seen. Myringotomy was performed after topical anesthesia had been induced with a 10 per cent cocaine hydrochloride solution. The returns were purulosanguinous. Culture of the pus yielded a growth of acid-fast bacilli on October 30. On October 10 roentgen examination of the mastoid process had revealed slight haziness of the cells of the posterior part of the mastoid process and of the cells in the region of the mastoid antrum. Administration of streptomycin (2 Gm. daily, intramuscularly, in divided doses) was started on October 31. The use of streptomycin was discontinued on December 27. Weekly examinations were performed. No discharge was noted after November 20. On December 3 there was no evidence of perforation. On December 30 the results of hearing tests were as follows: Right ear—whispered voice, 10/15; low conversational voice, 20/20; audiometric loss, 14 decibels (all ranges involved but especially the high range). Left ear—whispered voice, 15/15; low conversational voices, 20/20; audiometric findings, normal.

The patient had been transferred to Fitzsimons General Hospital on April 16, 1945, with the following diagnosis: (1) tuberculosis of the upper lobe of the right lung and the upper lobe of the left lung, reinfection type, chronic, active, far advanced; (2) pneumothorax of the right side, therapeutic, induced April 22, 1945; (3) effusion, pleural, right, secondary to (1) and (2). At this hospital the patient was noted to have thoracic empyema on the right side caused by mixed tuberculous and pyogenic infections. The patient then underwent a four stage thoracoplasty with removal of ribs 1 to 10. No empyema was noted as of March 1947. That condition was listed as cured. The infiltration of the left lung was found to be unchanged in appearance.

The final diagnosis was acute tuberculous otitis media, apparently healed.

Case 10.—H. K., a 33 year old enlisted man, was seen for the first time in the eye, ear, nose and throat clinic on June 4, 1946. He complained of ringing of the right ear, dizzy spells, falling to the right and a foul discharge of the right external auditory canal, unassociated with pain, since March 1945. Examination revealed that a moderate amount of foul yellowish material was being discharged into the right external canal. After this material had been thoroughly removed, a large central perforation of the right drum was noted. There was no nystagmus or spontaneous past pointing, no tenderness of the mastoid process and no facial paralysis. Hearing of the right ear was nil to loud spoken voice. The left ear was normal. Audiometric examination was not done at that time.

There was no essential change in the aural condition until the institution of streptomycin therapy, Dec. 31, 1946. Culture of the material discharged from the drum on November 7 gave a growth of the tubercle bacillus.

On December 31 administration of streptomycin was started (2 Gm. daily, in divided doses, intramuscularly). On Jan. 2, 1947 an audiogram revealed no loss of decibels for the left ear and an average loss of 68 decibels for the right ear. All frequencies were equally involved.

By February 13 the discharge had completely ceased. However, the large central perforation still persisted. On February 27 examination revealed that the canal of the external right ear was dry and that the drum of the right ear was intact. (An audiogram taken February 27 presented essentially the same

decibel losses as previously noted.) There has been no discharge of the right ear from the last-named date to the time of writing.

The initial roentgenogram of the chest revealed pneumothorax on the left side, a cavity of the left midlung, 2 cm. in diameter, and a small cavity of the medial portion of the apical region of the right lung. Streptomycin therapy was started on Dec. 31, 1946. One month later there had been considerable decrease in the size of the previously noted cavities. Two months later no change was noted.

Weekly smears of concentrated sputum showed acid-fast bacilli, and weekly cultures showed tubercle bacilli except for the one of March 4, 1947.

The final diagnosis was chronic tuberculous otitis media of the right side, apparently healed. The patient is still being treated with streptomycin at the time of writing.

CASE 11.—C. P. L., a 30 year old soldier, was seen for the first time in the eye, ear, nose and throat clinic on June 27, 1946. He complained of a purulent discharge of his left ear, unassociated with pain or acute infection of the upper respiratory tract. The examination of the ears, the nose and the throat gave essentially negative results except for the findings in the left ear. The external canal of the left ear presented a moderate amount of mucopurulent material, and there was a large anterior central perforation of the left drum. A culture of the material discharged showed a growth of tubercle bacilli. The patient received intramuscular injections of streptomycin (1.8 Gm. daily) and ear drops of streptomycin solution (120,000 units per cubic centimeter) from June 18 to October 18. Weekly examinations during that time showed no improvement. Ninety-six days after the institution of this therapy, culture of the material discharged from the ear showed a growth of tubercle bacilli. A laboratory study of the sensitivity of these organisms showed that they grew in concentrations of streptomycin greater than 10 units per cubic centimeter.

The results of the initial tests of hearing were: Right ear: whispered voice, 15/15; low conversational voice, 20/20; there was no decibel loss. Left ear: whispered voice, 2/15; low conversational voice, 5/20; audiometric loss, 30 decibels (conductive and perceptive type). The results of the final tests of hearing were essentially the same.

The initial roentgenogram revealed multilocular cavitations containing fluid in the upper lobe of the left lung. Streptomycin therapy was started June 18, 1946. One month later a roentgenogram revealed that the fluid in the cavities of the upper lobe of the left lung had disappeared. Two months later roentgenograms revealed a decrease in the size of the larger cavities in the upper lobe of the left lung. Three months later no change was noted. Weekly smears and cultures of concentrated sputum showed acid-fast or tubercle bacilli.

The final diagnosis was chronic suppurative tuberculous otitis media. The results of therapy indicate no improvement.

TUBERCULOSIS OF THE MASTOID PROCESSES

There is 1 case—with apparent healing.

CASE 12.—W. S., a 19 year old soldier, was hospitalized for the first time on Aug. 4, 1946, in Germany, where he was treated for bilateral otitis media. There he was given a course of penicillin therapy without effect. He was transferred to the United States and arrived at Fitzsimons General Hospital on September 9. On the same day he was seen for the first time in the eye, ear, nose and throat clinic.

The examination of the ears, the nose and the throat revealed essentially normal conditions except for pathologic changes pertaining to both ears. There was marked narrowing of the external canals, almost completely occluding them. The drums could not be seen. There was a slight amount of foul-smelling mucopurulent material coming from both canals. Both auricles revealed tenderness on being manipulated in every direction. There was no tenderness of the mastoid process, facial paralysis or nystagmus. Hearing was nil bilaterally for loud conversational voice located within 5 cm. (2 inches) of the ears. An audiogram revealed a loss of 93 decibels in the right ear and a loss of 94 decibels in the left ear, no tones being heard above that of 512 cycles per second. Roentgenograms of the mastoid processes taken on September 9 revealed marked sclerosis of both mastoid processes with probable cholesteatoma of the right process. The initial roentgenogram of the chest, taken September 9, revealed irregular linear infiltration of the upper lobe of the right lung, confluent in the apical region, and minimal infiltration of the apical region of the left lung. Initially the patient was treated by continuous application of wet dressings to both ears and was given penicillin. There was no notable improvement in the condition of the ears except for slight subsidence of the pain felt on manipulation of the auricles.

On October 17 the patient was observed to have paralysis of the left side of the face. Simple mastoidectomy was performed on the left side the same day. The entire mastoid area was necrotic and contained a large amount of creamy yellow pus. The entire dural plate was necrotic and was removed. There was pus between the plate and the dura, and the dura was thickened and covered with irregular grayish granulations. These were not touched. The outer third of the posterior canal wall was necrotic and was removed. The zygomatic cells were also necrotic. The bony plate of the lateral sinus was intact. The antrum was enlarged. The horizontal and posterior semicircular canals were bared and found to be intact. The wound was left wide open and packed lightly with plain gauze tape. Specimens were taken for smear and culture, and pieces of necrotic tissue were sent to the pathology laboratory for study. Smear and culture showed acid-fast bacilli and tubercle bacilli, and the pathologic report indicated that tubercules and acid-fast bacilli had been demonstrated after use of a technic of staining acid-fast bacilli.

Local as well as intramuscular administration of streptomycin was started on November 1; 2 Gm. of streptomycin was given intramuscularly, and 0.5 Gm. locally, daily. The mastoidectomy wound was packed in the following manner: A middle-sized rubber tube into which several holes had been bored was put into the mastoidectomy cavity and was surrounded with loosely packed plain gauze. Streptomycin was injected into the tube daily, whence it impregnated the surrounding gauze.

It was decided to perform simple mastoidectomy on the right side and to pack the mastoidectomy cavity in a manner similar to that just described, and inject streptomycin into the tube. This was done on November 5. The entire mastoid area was found to be composed of soft, necrotic diploic cells. The roof of the epitympanic cavity was found to be necrotic and was removed. The antrum was enlarged, and the posterosuperior bridge was removed. Smears and cultures and studies of the necrotic tissue taken from the pus and debris of the right mastoid process revealed acid-fast bacilli and tubercles in large numbers and also tubercle bacilli. Local treatment with streptomycin was continued bilaterally as described until Feb. 3, 1947. The mastoidectomy cavities were

repacked weekly in the manner recorded in the foregoing paragraph. The facial paralysis cleared up within a week after the first operation. After the second postoperative week, the mastoidectomy wounds became clean, and smears and cultures no longer showed tubercle bacilli. Both cavities gradually became filled with healthy granulation tissue, so that by February 3 there was no room for any further packing, and local application of streptomycin was discontinued. On February 7 secondary closures of both postauricular wounds were performed. The wounds had healed uneventfully by February 14.

About that time the patient began to hear sounds in his right ear, such as the closing of a door and the falling of a pan on the floor. At the time of writing, April 4, 1947, he can hear a moderate conversational voice approximately 120 cm. (4 feet) from the right ear and approximately 30 cm. (1 foot) from the left ear. Both external canals are normal in appearance and patent, with the exception of a slight prolapse of the roof of the left canal. Both drums are intact and essentially normal in appearance. Both mastoidectomy scars are well healed. The patient has gained approximately 13.5 Kg. (30 pounds).

The initial roentgenogram of the chest, Sept. 9, 1946, revealed, as previously noted, irregular linear infiltration of the upper lobe of the right lung, confluent in the apical region, and minimal infiltration of the apex of the left lung. Streptomycin was started on November 1. On Jan. 9, 1947 a slight regression of the infiltration of the upper lobe of the right lung was noted. On February 8 that finding had not changed. The latest roentgenogram of the chest, taken April 4, revealed only minimal involvement of the apex of the right lung.

Weekly smears and cultures of concentrated sputum were negative for acid-fast bacilli fifteen times in succession except for one positive twenty-four hour culture of concentrated sputum made October 17. Intramuscular injections of streptomycin were discontinued on April 3, 1947.

The final diagnosis was bilateral chronic tuberculous mastoiditis complicated with paralysis of the left side of the face and epidural abscess of the left side. The results of the final tests of hearing were: Moderate spoken voice: right ear, 4/20; left ear, 1/20. Audiogram: right ear, loss of 64 decibels; left ear, loss of 91 decibels (conductive and perceptive in both ears). The therapy has resulted in healing of the areas of disease.

PREPARATION AND ADMINISTRATION OF STREPTOMYCIN (PROCEDURES USED AT FITZSIMONS GENERAL HOSPITAL)

Procedures for Intramuscular Injection.—One gram (1,000,000 units) of streptomycin is diluted with 9 cc. of sterile water or sterile isotonic sodium chloride solution, making the final concentration 100,000 units per cubic centimeter.

The usual daily dose is 1.8 Gm. of streptomycin. The patient receives 300,000 units every four hours (day and night). The streptomycin is injected either in the deltoid region or in the buttock.

Procaine hydrochloride (2 per cent) has been added to the streptomycin solution just before the injection. However, there does not seem to be any appreciable difference as regards pain at the site of injection whether the streptomycin is injected with or without procaine.

Procedure for Nebulization.—One gram (1,000,000 units) of streptomycin is diluted with 19 cc. of sterile water, and a few drops of glycerin are added, making the concentration of the solution 50,000 units per cubic centimeter. (If the strength of the solution is made to exceed 400,000 units per cubic centimeter, the streptomycin will crystallize on the walls of the nebulizer.)

A Curry type nebulizer has been used at this hospital. By means of a rubber tube connected with an oxygen tank or wall outlet, oxygen is allowed to run into the nebulizer at the rate of 4 to 5 liters a minute.

The patient usually receives 0.5 Gm. of streptomycin daily by the nebulization route. From 2 to 3 cc. of solution (100,000 to 150,000 units) is put into the nebulizer, and then the oxygen is allowed to enter the nebulizer. It usually takes about fifteen minutes for the complete solution to be inhaled. The procedure described is repeated every four hours until the total daily amount of streptomycin has been given.

COMMENT

As we reviewed the cases reported in the foregoing pages the following impressions were received:

1. Streptomycin is undoubtedly a powerful therapeutic agent in the treatment of tuberculosis of the ear, the nose or the throat.

2. Regarding the larynx and the pharynx:

(*a*) All lesions disappeared and there was a return to apparent normalcy of both the pharynx and the larynx (cases 1, 2, 3, 4 and 7).

(*b*) Hoarseness disappeared (in all 7 cases).

(*c*) There was early disappearance of symptoms, such as dysphagia, sore throat, and pain on talking (in all cases).

(*d*) Apparent healing was achieved even though smears and cultures continued to show acid-fast bacilli or tubercle bacilli, respectively, and thoracic lesions were far advanced.

(*e*) Apparent healing was induced by either the nebulization or the intramuscular route of therapy, although a combination of both routes appears to be the treatment of choice (as illustrated in case 5).

(*f*) We have not been able to determine for what period the laryngeal and pharyngeal lesions and the symptoms are controlled in the presence of far advanced thoracic lesions, since our patients are still receiving streptomycin therapy or have been transferred recently to other hospitals.

3. Regarding the treatment of tuberculosis of the middle ears and mastoid processes, we have been much impressed with the results in cases 9, 10 and 12. In all 3 cases there is at the time of writing no evidence of tuberculous infection of the ears. In case 11 streptomycin had no effect clinically. In this case the tubercle bacilli were shown, by a laboratory study of their sensitivity, to grow in concentrations of streptomycin greater than 10 units per cubic centimeter. This in the opinion of the laboratory chief represented a resistant organism.

4. Regarding case 8, in which septal perforation was observed, we feel that the patient was under our observation too short a period

for us to determine the full effect of streptomycin. Local packs exerted a definite beneficial effect. It would appear, however, that intramuscular injection of streptomycin in addition to local packing would be the treatment of choice in this type of case.

5. It is interesting to note that there were no toxic symptoms in any of the cases except for slight dizziness—never severe enough for spontaneous complaint on the part of the patients.

SUMMARY

This paper deals with 12 patients at Fitzsimons General Hospital who were treated with streptomycin for tuberculosis of the ear, the nose or the throat.

The breakdown of the results is as follows:

1. Tuberculosis of the larynx—6 patients, 4 with apparent healing and 2 with improvement.
2. Tuberculosis of the pharynx and the larynx—1 patient, with apparent healing.
3. Tuberculosis of the nasal septum—1 patient, with improvement.
4. Tuberculosis of the middle ears—3 patients, 2 with apparent healing and 1 without improvement.
5. Tuberculosis of the mastoid processes—1 patient, with apparent healing.

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LYMPHOID EUSTACHIAN SALPINGITIS: ITS EFFECT ON TUBAL PATENCY

Selective Criteria for Nasopharyngeal Irradiation

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LYMPHOID eustachian salpingitis, or lymphoid tubotympanitis, may be defined as any lymphoid hyperplasia in or about the eustachian tube which interferes with the physiologic opening of the tube and produces symptoms referable to the middle ear. It is usually associated with a chronic catarrhal inflammatory process.

Since the work of Crowe and Baylor,[1] this entity has been widely treated by the nasopharyngeal application of radium or radon. As shown in figure 1 and tabulated by Boies,[2] the radium applicators deliver a maximum dose on contact and an ineffectual minimum of radiation at a distance of 5 mm. Therefore, they can be of primary value only in the treatment of disease occurring in the immediate neighborhood of the pharyngeal orifice of the eustachian tube. Obstruction in the peripheral portion can be more advantageously treated with high voltage roentgen rays.

If radiation is to be used judiciously in the treatment of lymphoid eustachian salpingitis, the position of the obstruction in the eustachian tube must be known. The obstruction may be (1) central or (2) peripheral (fig. 2). Central obstruction, at the pharyngeal orifice of the eustachian tube, will receive maximum benefit from the nasopharyngeal application of radium. Peripheral obstruction is beyond the effective range of the radiations of the nasopharyngeal applicator. In this study, the quantitative degree of obstruction of the eustachian tube has been measured with the mercurial manometer; first, the number of millimeters of mercury pressure required for the patient to autoinflate the middle ear is obtained (the degree of central obstruction);

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1. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112**:585-590 (Feb. 18) 1939.

2. Boies, L. R.: Irradiation of Nasopharyngeal Lymphoid Tissue: An Evaluation, Arch. Otolaryng. **44**:129-140 (Aug.) 1946.

second, the number of millimeters of mercury pressure required to inflate the middle ear through the eustachian catheter is obtained (the degree of peripheral obstruction).

APPARATUS

The apparatus used (fig. 3) to determine quantitatively the patency of the eustachian tube consists of the mercurial manometer of a sphygmomanometer, attached by one arm of a Y tube to a Politzer bag and by the other arm of the Y tube to rubber tubing which may be adapted either to an olivary nasal tip or to a eustachian catheter.

PROCEDURE

To determine the degree of central tubal obstruction quantitatively, a procedure similar to that described by Perlman [3] and Hartman [4] has been used. The olivary nasal tip is attached to the mercurial manometer. The patient places the nasal

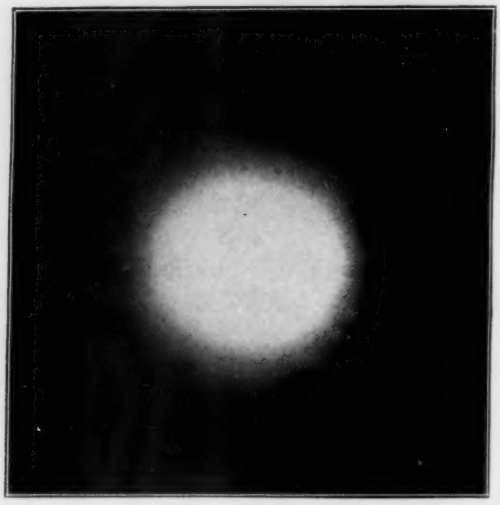

Fig. 1.—This exposure of the nasopharyngeal radium applicator demonstrates the localized effect of the radium emanations, the potency of the radiations being inversely proportional to the square of the distance. The advantage of radium is this localized effect. However, if the disease is not localized, high voltage roentgen radiation is indicated.

tip in one nostril, occludes the opposite nostril and performs the Valsalva maneuver, blowing through the nasal tip against the column of mercury. The manometric reading is taken at the moment air escapes into the middle ear. This moment is determined objectively with the auscultating tube or by direct observation of the tympanic membrane. Perlman [3] and Hartman [4] observed that when the Valsalva maneuver is performed the eustachian tube will normally open at 20 to 40 mm. of mercury. In this paper the pressure obtained by this procedure is called the "Valsalva inflation pressure." The procedure was repeated three

3. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

4. Hartman, A.: Experimentelle Studien über die Funktion der eustachischen Röhre, Leipzig, Veit & Co., 1879.

times with each patient, the lowest pressure obtained being recorded as the Valsalva inflation pressure.

Peripheral tubal patency is determined quantitatively by passing the eustachian catheter through the pharyngeal orifice and measuring the pressure required to inflate the middle ear through the catheter. To prevent muscular spasm, the pharyngeal orifice of the eustachian tube is anesthetized with a 10 per cent cocaine hydrochloride solution containing epinephrine hydrochloride in the concentration of 1 : 5,000. The eustachian catheter is inserted as in routine practice and adapted to the apparatus. Gentle pressure is then created by compression of the Politzer bag. Normally, with the catheter in position, extending 4 to 5 mm. into the lumen, little pressure is required to separate the collapsed walls of the eustachian tube and inflate the middle ear. Again, the moment of inflation of the middle ear is determined with the diagnostic tube. The lowest of three readings is the figure recorded in this study. With this apparatus, to 6 mm. of mercury is considered the normal "catheterization inflation pressure" for practical purposes. The actual normal catheterization inflation pressure is lower and

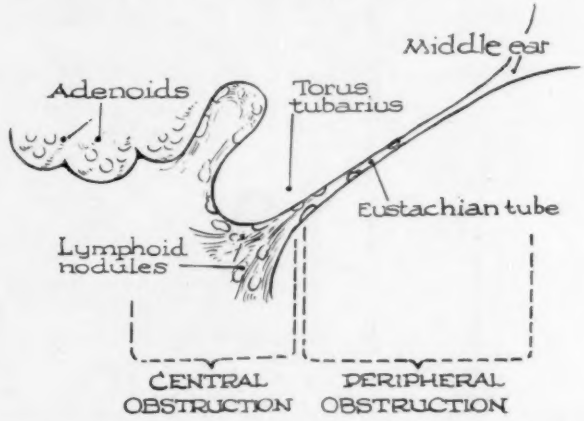

Fig. 2.—This diagram of a longitudinal section of the eustachian tube, taken through the torus tubarius, demonstrates central obstruction, at the pharyngeal orifice, in contrast with peripheral obstruction. Only central obstruction is within the effective range of the radiations of the nasopharyngeal radium applicator.

can be obtained only with the less practical water manometer or pressure chamber. The catheterization inflation pressure thus determines the patency of that portion of the eustachian tube which is beyond the effective range of radium or radon applied to the nasopharynx.

PATIENTS STUDIED

In this study the Valsalva inflation pressure and the catheterization inflation pressure have been determined for 100 diseased eustachian tubes. The patients were all males, with an average age of 27.5 years. They all had long-standing complaints of well established recurrent tubal obstruction, recurrent otitis media, middle ear deafness or aero-otitis media. The tympanic membrane was moderately or greatly retracted. The moderate or pronounced hypertrophied lymphoid tissue on the lateral wall of the nasopharynx covered the torus tubarius or extended into the pharyngeal orifice of the tube. Routine complete otoscopic, rhinoscopic,

nasopharyngoscopic and audiometric examinations revealed nothing which has not been previously described (Farrior [5]).

TUBAL PATENCY

The catheterization inflation pressure was normal in 61 per cent of the entire group of 100 eustachian tubes (table, group A). In these, the values averaged only 3.4 mm. of mercury, an average which reveals no evidence of significant peripheral obstruction of the eustachian

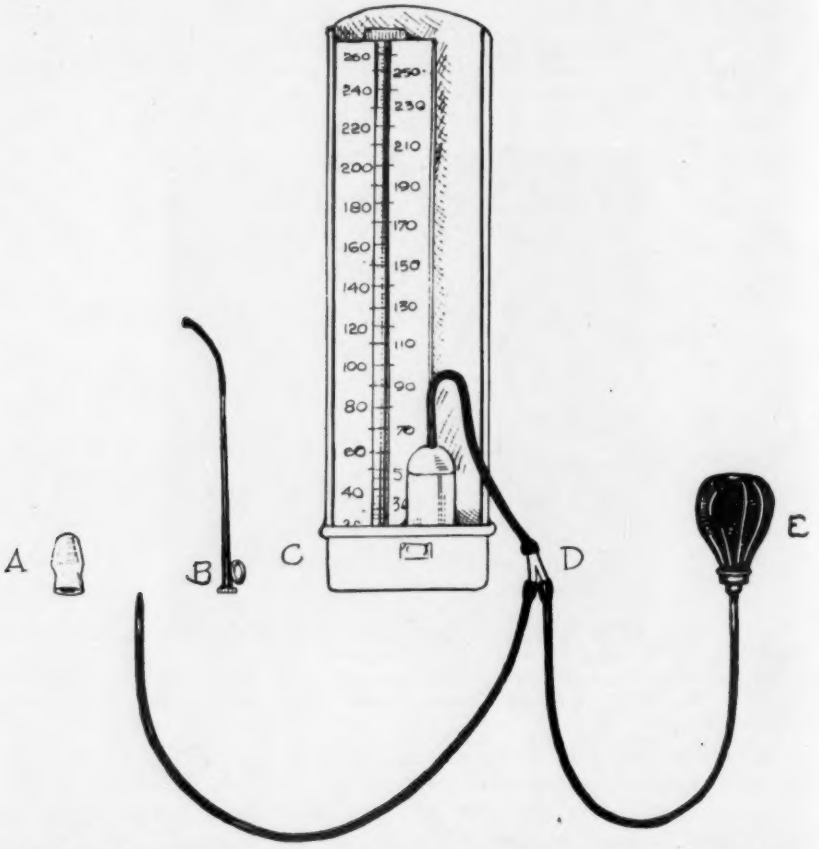

Fig. 3.—The tubal pressure apparatus consists of: *A*, olivary nasal tip; *B*, eustachian catheter; *C*, mercurial manometer; *D*, Y connecting tube; *E*, politzer bag.

tube; in 46 (75.4 per cent) of these it was not possible to autoinflate the middle ear by the Valsalva maneuver, even though the maximal pressures produced averaged 77.2 mm. of mercury. In the remaining 15 (24.6 per cent) of this group there was demonstrated evidence of obstruction of the pharyngeal orifice of the eustachian tube, the

5. Farrior, J. B.: Histopathologic Considerations in the Treatment of the Eustachian Tube, Arch. Otolaryng. **37**:609-621 (May) 1943.

elevated Valsalva inflation pressures averaging 51.6 mm. of mercury. Therefore, in 61 per cent of the entire group the only obstruction in the eustachian tube was at the pharyngeal orifice. Such cases, with pathologic changes only at the pharyngeal orifice of the eustachian tube, are excellent for the nasopharyngeal radium applicator.

The catheterization inflation pressure was elevated to 8 or 10 mm. of mercury in 15 eustachian tubes (table, group B). In 13 (86.6 per cent) of these it was not possible to autoinflate the middle ear, even though the maximal pressures by the Valsalva maneuver averaged 92.2 mm. of mercury. The 2 patients who could autoinflate the middle ear required an average Valsalva inflation pressure of 55 mm. of mercury. This indicates that in 15 per cent of the 100 eustachian tubes studied there was mild obstruction peripheral to the pharyngeal

Tubal Pressure Changes in 100 Eustachian Tubes with Lymphoid Eustachian Salpingitis

	Eustachian Tubes	Percentage in Which Valsalva Maneuvers Were Unsuccessful	Percentage in Which Valsalva Inflations Were Successful (Pressures Above Normal)
Group A			
Normal catheterization inflation pressures (2 to 6 mm. of mercury).....	61	75.4	24.6
Group B			
Moderately elevated catheterization inflation pressures (8 to 10 mm. of mercury).....	15	86.6	13.4
Group C			
Markedly elevated catheterization inflation pressures (10 to 50 mm. of mercury).....	24	91.7	8.3
Total.....	100	81.0	19.0

orifice. In such tubes the primary pathologic condition is at the pharyngeal orifice, within reach of the nasopharyngeal radium applicator; however, better results can be expected if nasopharyngeal application of radium is supplemented with high voltage radiation directed to the peripheral portion of the tube.

The catheterization inflation pressure was greater than 10 mm. of mercury in 24 of the diseased eustachian tubes (table, group C). The values ranged from 12 to 50 mm. of mercury, with an average of 25.8 mm. of mercury. In 22 (91.7 per cent) it was not possible to autoinflate the middle ear, even though the maximal pressures of the Valsalva maneuver averaged 78.2 mm. of mercury. Two patients (8.3 per cent) could autoinflate the eustachian tube and exhibited elevated Valsalva inflation pressures averaging 54.0 mm. of mercury. In these 24 cases, in addition to the obstruction at the pharyngeal orifice, there was great obstruction in the peripheral portion of the tube, which could be reached only by high voltage roentgen radiation.

Of the 100 eustachian tubes studied, 81 could not be opened by the Valsalva maneuver. The remaining 19 opened with a high average pressure. In the 81 eustachian tubes which could not be opened by autoinflation, the maximum pressures produced averaged 79.8 mm. of mercury. The maximum pressures ranged from 40 to 140 mm. of mercury, the height of the maximum pressure being dependent on the muscular powers of the patient. In none of these cases was the patency of the eustachian tube altered by nasal shrinkage obtained with a spray of 1 per cent ephedrine hydrochloride in isotonic solution of sodium chloride or by the topical application of a 10 per cent cocaine hydrochloride solution with epinephrine to the pharyngeal orifice of the tube. In only 19 cases was the patient able to autoinflate the middle ear. These required an average Valsalva inflation pressure of 52.2 mm. of mercury.

PERIPHERAL EUSTACHIAN OBSTRUCTION

The interpretation of the elevated catheterization inflation pressures must be on the basis of histopathologic knowledge (Farrior [5]). All these patients (group C) exhibited evidence of pathologic narrowing of the lumen of the eustachian tube peripheral to the pharyngeal orifice. This pathologic narrowing may be the result of the development of lymphoid nodules or of active chronic inflammation, or it may be produced by cicatricial constriction of the lumen and adhesive bands, the end result of an earlier inflammatory process. Auscultation with the diagnostic tube may be of value in interpreting the lesion present.

Auscultation revealed mucus within the eustachian tube in 11 ears (45.8 per cent of group C) with elevated catheterization inflation pressures. When the mucus was cleared away, the inflation pressure remained elevated. It is assumed that in this group the obstruction is in part secondary to an active inflammatory process or the presence of a lymphoid nodule within the eustachian tube and that there is hope of improvement if therapy can produce resolution of the disease.

Auscultation revealed a dry eustachian tube with inflation producing a high-pitched squeaking whistle in 13 ears (54.2 per cent of group C) with elevated catheterization inflation pressures. It is assumed that in this group the obstruction is produced by cicatricial tissue, the end result of a preexisting inflammatory process, and that no known form of therapy can promote resolution.

Owing to the long-standing lymphoid eustachian salpingitis and the concomitant chronic catarrhal inflammation in this adult age group, there is a higher incidence of peripheral tubal obstruction than one would expect to find in children. However, peripheral tubal obstruction accounts for some of the failures of nasopharyngeal irradiation in children.

COMMENT

In cases of disease of the middle ear and deafness with lymphoid hypertrophy about the pharyngeal orifice of the eustachian tube, the point of greatest obstruction of the tube is at the pharyngeal orifice, where it may receive the maximum benefit from nasopharyngeal application of radium or radon. With due appreciation of the complex physiology of the opening of the eustachian tube, the comparison of the patient's ability to autoinflate the middle ear with the pressure required to inflate the middle ear through a eustachian catheter represents a practical method of determining the location of disease within the eustachian tube.

CLINICAL APPLICATION

If there is lymphoid hypertrophy at the pharyngeal orifice of the eustachian tube but the middle ear inflates easily by catheterization, maximum benefit may be expected from the use of the nasopharyngeal radium applicator alone. These ears will also inflate easily by politzerization.

If there is lymphoid hypertrophy at the pharyngeal orifice of the eustachian tube and increased pressure is required to inflate the middle ear with the eustachian catheter, the nasopharyngeal application of radium should be supplemented with, or replaced by, high voltage roentgen irradiation along the course of the eustachian tube. Ears thus involved are difficult to inflate by politzerization.

In other words, if, in the presence of lymphoid eustachian salpingitis, the middle ear cannot be easily inflated by politzerization, there is probably obstruction in the peripheral portion of the tube, and nasopharyngeal radium therapy should be supplemented with, or replaced by, high voltage roentgen irradiation along the course of the eustachian tube. Politzerization can be satisfactorily employed in most children.

DIFFERENTIAL DIAGNOSIS

Persistently Patent Eustachian Tube.—The persistently patent eustachian tube can easily be differentiated from lymphoid eustachian salpingitis by having the patient autoinflate the middle ear by the Valsalva maneuver, with or without the mercurial manometer. I have recently examined 3 patients with abnormally patent eustachian tubes. These patients were able to autoinflate the middle ear with pressures of 4, 6 and 10 mm. of mercury, respectively. On catheterization the air entered the middle ear with a blowing sound at a pressure of 2 mm. of mercury. Two of these patients had previously been indiscriminately treated with the nasopharyngeal radium applicator.

Otosclerosis.—In uncomplicated otosclerosis the Valsalva inflation pressure and the catheterization inflation pressure are within the limits of normal. When there is superimposed lymphoid eustachian sal-

pingitis and studies show altered tubal patency, irradiation is indicated in an effort to maintain the hearing at the patient's optimum level. However, patients with otosclerosis and studies showing normal tubal patency are frequently unjustifiably treated with the nasopharyngeal radium applicator.

Nerve Deafness.—In uncomplicated nerve deafness the Valsalva inflation pressure and the catheterization inflation pressure are within the limits of normal. When there is superimposed lymphoid eustachian salpingitis and studies reveal altered tubal patency, irradiation is justified in an effort to maintain the hearing at the patient's optimum level. However, patients with nerve deafness and studies showing normal tubal patency are frequently unjustifiably treated with the nasopharyngeal radium applicator.

SUMMARY

"Lymphoid eustachian salpingitis" or "lymphoid tubotympanitis" is the specific name for a basic pathologic condition, and this condition must be differentiated from other forms of disease of the eustachian tube.

An apparatus is described for the measurement of the patency of the eustachian tube.

One hundred diseased eustachian tubes, exhibiting moderate to severe lymphoid hypertrophy about the pharyngeal orifice associated with extensive changes in the tympanic membrane, have been studied. The pressure in millimeters of mercury required for the patient to autoinflate the middle ear has been compared with the pressure required to inflate the middle ear through the eustachian catheter.

In 81 per cent of the eustachian tubes with lymphoid hypertrophy about the pharyngeal orifice it was not possible to autoinflate the middle ear by Valsalva's maneuver.

Examination revealed no evidence of obstruction peripheral to the pharyngeal orifice in 61 per cent, mild obstruction peripheral to the pharyngeal orifice in 15 per cent and moderate or considerable obstruction peripheral to the pharyngeal orifice in 24 per cent.

The position of the existing pathologic condition determines the type of radiation therapy to be employed.

A lymphoid obstruction at the pharyngeal orifice of the eustachian tube is in position to receive maximum benefit from the superficial penetrations of the nasopharyngeal radium applicator.

A lymphoid obstruction in the peripheral portion of the eustachian tube will receive little benefit from the superficial penetrations of the nasopharyngeal radium applicator and should be treated with high voltage roentgen rays.

The nesopharyngeal radium applicator is indicated only when there is objective evidence of lymphoid hyperplasia and demonstrable impairment of tubual patency.

910 Citizen's Building.

ABSTRACT OF DISCUSSION

DR. GILBERT FISHER, Birmingham, Ala.: I consider it a distinct privilege to discuss this splendid paper. This topic is one in which I have been greatly interested for a number of years. I had the privilege and pleasure of working with Dr. S. J. Crowe when the program for irradiation of hyperplastic nasopharyngeal lymphoid tissue was first introduced in the Johns Hopkins Hospital.

Since 1940 we have examined and treated with radiation approximately 2,000 persons who had definite high tone loss because of obstruction to the eustachian tubes. The results obtained with this type of treatment in well selected cases have been uniformly good. We have found that in approximately 3 per cent the conduction hearing has not returned entirely to normal after irradiation of the obstructed tube, even though the lymphoid tissue which prior to treatment covered the orifices of the eustachian tube has diminished to a point where one visualizes an open tubal orifice.

When we find a residual hearing loss in young persons who have been treated with radiation and removal of the obstructing mass of lymphoid tissue from the orifice of the eustachian tube, we know that along the course of the eustachian tube there must be some obstruction caused either by fibrous adhesions or by small islands of lymphocytes present in the submucosa. Our routine procedure for these patients in the hearing clinic of the Medical College of Alabama is to make an audiometric study of each ear. Immediately after the audiometric study has been completed, a eustachian tube cannula is passed under direct vision aided by the electric nasopharyngoscope. A eustachian tube bougie is then passed, and if no resistance is encountered, the eustachian tube is inflated. A second audiogram is taken, and if the hearing is found to have been definitely improved, we then give a course of roentgen therapy directed toward the middle ear and along the course of the eustachian tube in an endeavor to produce atrophy in any submucosal masses of lymphoid cells. This roentgen therapy is usually given in the following manner: 150 kilovolts; filter, 0.5 mm. of copper and 1 mm. of aluminum; dosage, 150 roentgens to each side of the neck once weekly for six successive weeks.

One month after the roentgen therapy has been given, an audiogram is again taken to see whether definite improvement has followed this type of therapy.

We now feel absolutely certain that if the patients are well selected and treated, preferably prior to puberty, the hearing loss due to nasopharyngeal lymphoid tissue obstructing the orifices of the eustachian tube can be completely corrected, with reestablishment of tubal patency. However, if the eustachian tube has been obstructed long enough (a number of years) for adhesions to have formed within the middle ear or along the course of the eustachian tube, the prognosis for a return to normal hearing must be guarded.

It is our plea that all children of early school age have a complete examination of the ears, the nose and the throat, including use of the electric nasopharyngoscope, as well as careful audiometric studies in a relatively soundproof room. If this type of examination can be successfully and carefully carried out on all young school children, I believe, as does Dr. Crowe, that a high percentage of the conduction deafness now exhibited in the adult population could be completely eradicated in one or two generations.

Dr. Farrior has presented a wonderful series of cases and has established a most scientific group of eustachian tube pressure values which will be of great

use to all of us who are studying this problem. I heartily agree with his observations and with his methods of treatment, and I feel certain that this splendid presentation will add greatly to our diagnostic armamentarium.

Dr. J. R. Lindsay, Chicago: Dr. Farrior's investigations have been directed to an important field, since the diagnosis and treatment of disorders of the eustachian tube are of primary importance in the prevention of middle ear hearing impairment. His attempt to make an accurate objective determination of the site of tubal blockage is highly commendable.

Two of the questions which this report brings up for discussion are that of the reliability of the Valsalva test and the catheterization inflation pressure as indicators of tubal blockage and that of the interpretation of peripheral tubal blockage, that it may be due to a lymphoid nodule in the peripheral part of the tube and is therefore an indication for high voltage roentgen ray therapy.

The Valsalva test is of course not the physiologic method of opening the tubes, and the interpretations are subject to certain reservations. A few persons with normally functioning tubes are not able to inflate them by the Valsalva method. The pressure necessary to autoinflate the ears varies with the position of the body and the head. It is also characteristic that an acute or a subacute inflammatory episode in the throat, makes it difficult, or causes inability, to autoinflate the ears. These factors I assume have been taken into consideration by the essayist.

The pressure necessary to inflate the tube per catheter has been used as the indicator of peripherally located obstruction. Here again certain qualifications need to be mentioned. For example, a plug of tenacious exudate in the tube will increase the pressure necessary to inflate the ear by either of the methods under discussion until the exudate has been displaced or removed. Much difficulty may be experienced in expelling such a plug through a myringotomy incision, but the tube may inflate easily afterward. Such plugs of exudate often recur, especially during the winter season.

The author points out that one of the causes for peripheral tubal occlusion may be a lymphoid nodule, and it is on this basis I assume that high voltage roentgen ray therapy is recommended.

As far as I have been able to determine by examination of temporal bones and from published reports, the evidence to indicate that lymphoid tissue placed beyond the orifice of the tube plays any important part in peripheral tubal obstruction is not convincing. A few microscopic nodules of lymphoid tissue have been seen in the mucosa lining the tube but these could hardly be interpreted as a cause of blockage. Only rarely have these contained a germinal center. Glandular tissue is the most abundant element in the lining of the tube.

There are many descriptions available, among them Dr. Farrior's report of several years ago, showing the effect of inflammatory processes on the mucosa of the tube and middle ear. It appears that such inflammatory changes are more likely to be the important factor in peripherally located tubal obstruction. This is especially so in those cases in which there is visible evidence of adhesive processes in the middle ear.

In dealing with this type of case, my associates and I have observed that the removal of the predisposing factor at the pharyngeal orifice, whether infection, presence of lymphoid tissue or allergic reaction, plus the elimination of exudate or serous secretion in the middle ear has been the effective part of the therapy. The addition of high voltage roentgen therapy has been usually of little definite value.

On the basis of existing pathologic evidence and our own clinical results we believe that the indications for high voltage roentgen ray therapy in this type of case are somewhat meager.

I agree with the essayist's final point in his summary to the effect that a radium applicator should be used only in the presence of demonstrable impairment of tubal patency.

DR. STACY R. GUILD, Baltimore: I heartily agree with Dr. Farrior's conservative attitude in the use of the radium applicator. One of the greatest fears of our group at Baltimore ever since this work began has been that the applicator would be used indiscriminately. We have made this point over and over.

Furthermore, I agree with Dr. Lindsay's conservatism in saying that use of radium applicator is indicated only for nasopharyngeal lymphoid tissue. Like Dr. Lindsay, I have never seen any benefit from attempts to irradiate the peripheral part, as Dr. Farrior calls it, of the eustachian tube. Dr. Lindsay stated that germinal centers occur but rarely in the mucosa of the eustachian tube itself. I agree. In fact, I know of only 2 cases in our entire collection of sectioned ears that have true lymphoid nodules in the mucosa of the eustachian tube. Both are near the tympanic end, and both patients had had chronic suppurative otitis media for years. Otherwise, the lymphoid tissue in the subepithelial layer of the tube is of the diffuse type, not of the nodular type. Rarely, if ever, is a mitotic figure present in it. The dosages of radiation, whether of radium or of roentgen rays, which are used to treat this condition are in the limits that affect only mitosis; therefore no rational reason exists for irradiation of the so-called peripheral portion of the eustachian tube.

Occlusion of the peripheral, or osseous, portion of the eustachian tube, when it occurs without suppurative otitis, is in my opinion due usually to simple mucosal edema secondary to mechanical obstruction of the nasopharyngeal portion of the lymphatic vessels that drain the mucosa of the tube. These lymphatic vessels course from the tympanic to the pharyngeal end of the tube, then pass for a short distance in the mucosa of the nasopharynx before penetrating either the superior constrictor muscle or the buccopharyngeal membrane to reach the lateral retropharyngeal lymph glands. Hyperplastic lymphoid nodules near the pharyngeal orifice of the tube can thus readily interfere with lymphatic drainage of the entire tube, and the logical treatment for edema of any part of the tube would seem to be reduction of the size of the lymphoid masses in the nasopharynx.

On the whole, therefore, I would be even more conservative than is Dr. Farrior.

DR. J. BROWN FARRIOR: I want to thank the discussers for not having been too merciless in their discussion.

With due appreciation of the complex physiology of the eustachian tube and the simplicity of the Valsalva maneuver as a solution to the problem of opening the pharyngeal orifice, certainly one can conclude, first, that the Valsalva maneuver, with the head directed 30 degrees forward, will definitely eliminate from radiation therapy those with abnormally patent eustachian tubes.

Second, in the studies of Perlman, Hoagland and others, approximately 80 to 90 per cent of normal eustachian tubes could be opened by the Valsalva maneuver; although this maneuver is not foolproof in determining central obstruction, it is, from my studies, one of the best sources of evidence one has of existing central obstruction of the eustachian tube.

In catheterization, as far as there being an existing plug in the eustachian tube, in these studies both the Valsalva maneuver for the Valsalva inflation pressure and the catheterization inflation were performed three times each at the same examinations, the lowest pressure being recorded in the study.

I, too, experienced difficulty in my histopathologic studies of the eustachian tube in finding existing structures. Contrary to Dr. Guild's opinion that this peripheral obstruction is lymph stasis, it has been my impression that most of the peripheral obstruction was produced by cicatricial narrowing of the eustachian tube, in which no known form of therapy will produce a resolution.

In presenting this study it has been my effort to eliminate the radium applicator in those cases in which there was no existing tubular obstruction.

UNUSUAL PULMONARY COMPLICATIONS RESULTING FROM PROLONGED LODGING OF NONOPAQUE FOREIGN BODY IN LEFT MAIN STEM BRONCHUS

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DELAY in the diagnosis and the removal of a nonopaque foreign body from the bronchopulmonary tract may lead to severe and irreversible pulmonary damage. The case reported here serves well to illustrate the gamut of pulmonary complications which follow a prolonged sojourn of a nonopaque foreign body involving the left main stem bronchus.

REPORT OF CASE

The patient, a boy aged 10, was first seen by a general practitioner on Dec. 1, 1946. A brief history obtained from the parents revealed that several days previously he had been taken with sudden attacks of coughing and bronchial wheezing. Because these symptoms had increased in severity and there was now a marked elevation of temperature, he was sent to the local hospital to undergo roentgen examination of the chest. The radiologist reported moderate bilateral pulmonary infiltration suggesting bronchopneumonia (fig. 1 *A*). Penicillin and symptomatic therapy were immediately begun. Three days later, on December 3, general mediastinal and subcutaneous emphysema developed, involving the entire front and back of the chest and the fascial planes of the neck, and extending to the supraciliary regions. Roentgen examination of the chest confirmed this clinical finding (fig 1 *B* and *C*). The emphysematous condition lasted for a period of seven days.

In view of the patient's condition, which was becoming progressively worse, a roentgenogram of the chest was taken on December 14. It revealed obstructive emphysema of the entire left lung (fig. 1 *D*). In spite of this added and important evidence, still no thought was given to the possibility of a foreign body as the causative factor of the existing pulmonary condition. On Jan. 13, 1947, six weeks after the day of admission, a roentgen study of the chest revealed complete atelectasis of the left lung (fig. 2 *A*). During this period the daily range of the patient's temperature was from 99 to 105 F. On January 17 further roentgen studies revealed some formation of fluid in the left lower part of the chest. On account of the high temperature, it was felt that the fluid was purulent in type, and a thoracotomy with tube drainage was performed. Subsequent to this operation, the patient's condition took a greater turn for the worse. It was at this time that the attending physicians decided to seek bronchoscopic consultation. They telephoned me, and I told them of the possibility of a foreign body and advised that a bronchoscopic examination be made immediately. On January 18 the patient was admitted to the bronchoscopic department of the Ellis Hospital, where an emergency bronchoscopic examination revealed a for-

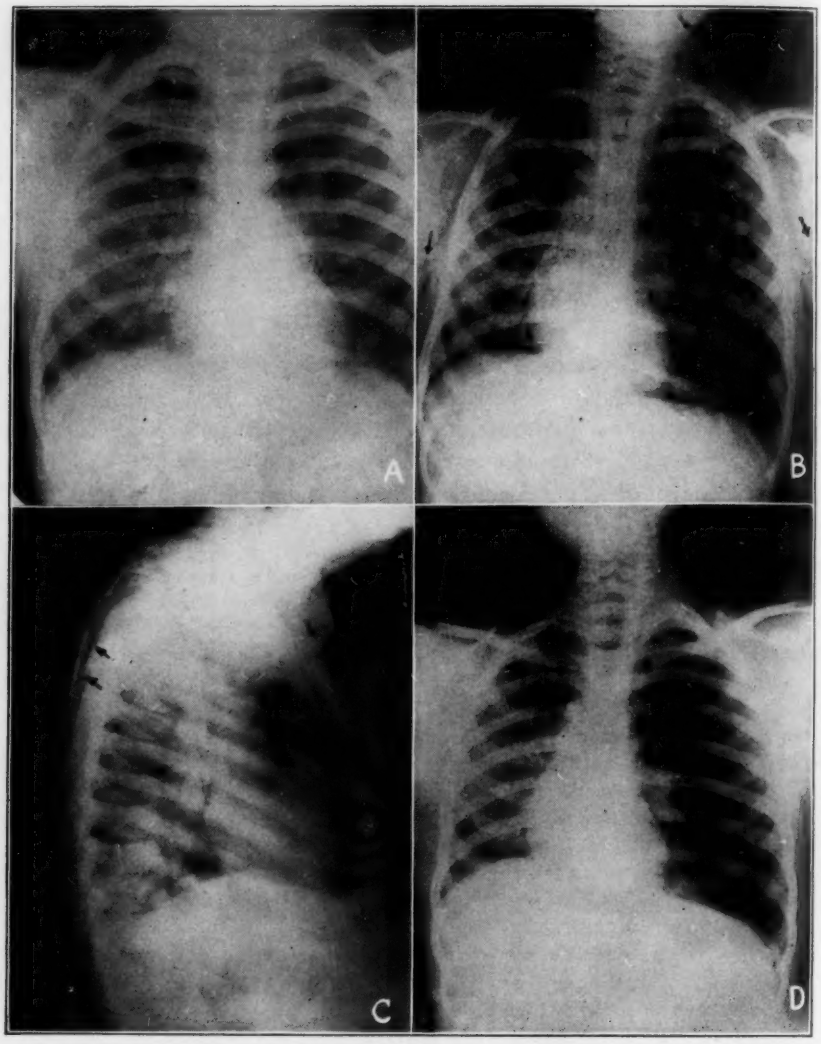

Fig. 1.—*A*, roentgenogram taken on the first day of hospitalization, December 1. It was interpreted as suggesting slight bronchopneumonia. It is not uncommon to see this type of pulmonary reaction early after the lodging of a nonopaque foreign body in the bronchial tract. The increase of bronchial secretion, due to the irritative action of the foreign body, usually results in a spilling over to the opposite side, causing the pulmonary reaction.

B and *C*, roentgenograms taken on December 3, showing diffuse emphysema of the chest and cervical regions, as indicated by arrows. This complication is seldom seen with smooth foreign bodies which have invaded the bronchial tract. A possible explanation of this unusual finding is that the violent coughing may have led to a slight rupture of the bronchial wall, allowing air to enter the interstitial tissues.

D, roentgenogram taken on December 14. It reveals obstructive emphysema of the left lung. Note the greater transparency on the obstructed side with depression of the diaphragm. If the ballooning of the lung is marked, the axis of the ribs is changed, with resultant widening of the intercostal spaces. An important finding in obstructive emphysema or expiratory check valve mechanism is the shifting of the cardiac shadow toward the uninvolved side, as shown by fluoroscopic examination.

eign body, the rubber sac of a fountain pen, completely obstructing the left main stem bronchus. Following its removal, the patient made an uneventful recovery and was discharged on the seventh day.

Nonopaque foreign bodies, particularly in children and infants, present a most troublesome problem in diagnosis, since in many instances they are frequently overlooked as a diagnostic possibility. It is only when severe pulmonary reaction has already taken place that these patients are sent to the bronchoscopist for examination. Unfortunately, because of an inadequate history and because of the fact that these foreign bodies are not visualized by roentgenographic study, many of these patients in whom pulmonary symptoms develop are at first treated for bronchitis, pneumonia or bronchial asthma.

In every instance in which a child or an infant seen for the first time is suffering from obvious pulmonary infection, a careful history, obtained from the parents, should include information regarding recent attacks of coughing and

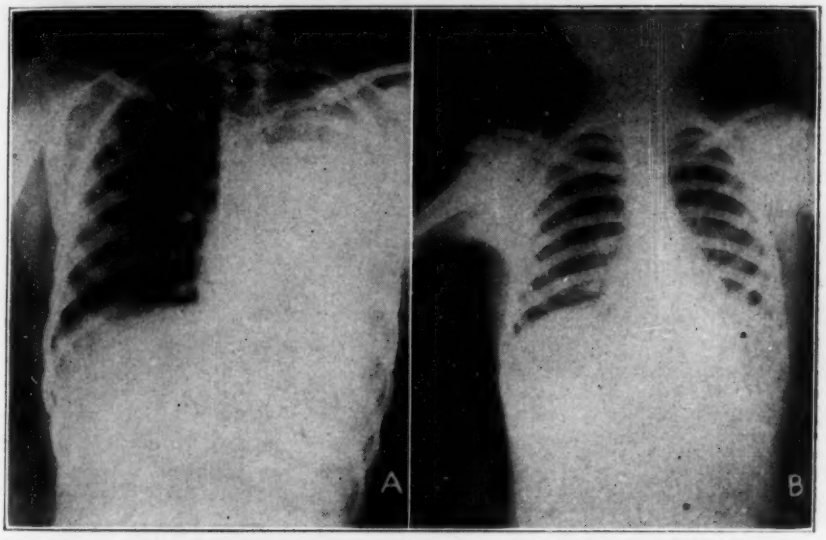

Fig. 2.—*A*, roentgenogram taken on January 13. It reveals complete atelectasis of the left lung. The mechanism, which previously was expiratory check valve in type, has now changed to that of a complete check valve, obstructing both the ingress and the egress of air. The roentgen observations now reveal marked density of the pulmonary area involved; the intercostal spaces are narrowed, the diaphragm is elevated and the mediastinal structures, because of the increased negative pleural pressure, are now displaced toward the affected side.

B, roentgenogram taken seven days after removal of the foreign body. It shows marked clearing of the atelectasis. A roentgenogram taken two weeks later, which is not shown in this article, revealed that the lung had regained its normal function completely.

choking while eating or drinking. If wheezing is present, special note should be made of the duration and the mode of onset. A careful history, thus obtained, may prove to be an important guide in the diagnosis of an aspirated foreign body. When in the case being reported the child was closely questioned, he remembered that several weeks before this he went to bed chewing on a piece of rubber.

The bronchial pulmonary tract is endowed with a powerful natural defensive mechanism. Even when obstructive atelectasis has existed for some time, as it had in the present case, the removal of the foreign body and aspiration of the retained secretions will in many instances restore normal function to the involved lung. However, this stage of complete bronchial obstruction, with possible subsequent irreversible pulmonary damage, might never be reached if every pediatrician and general practitioner of medicine would strongly consider the diagnostic possibility of an aspirated foreign body as the causative factor in any case in which a child or an infant is suffering from obvious bronchial and pulmonary infection.

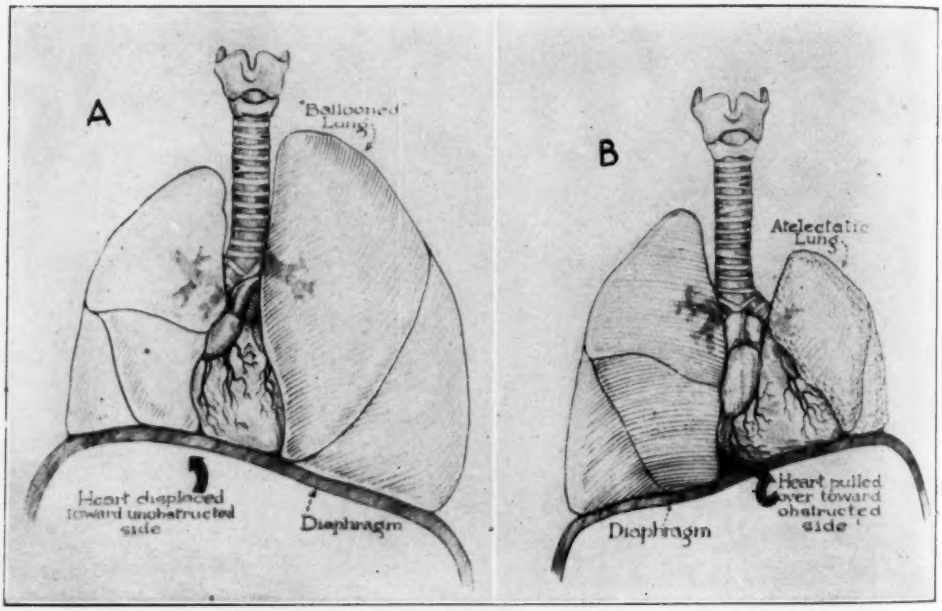

Fig. 3.—*A*, schematic drawing showing the relative disproportion of the ballooned or obstructed lung as compared with the uninvolved lung. This is the typical anatomic appearance of the lungs in a well developed case of obstructive emphysema.

B, schematic drawing illustrating the anatomic appearance of the lungs in a case of atelectasis of the left lung due to complete bronchial obstruction.

MECHANICS OF THE BRONCHIAL TRACT

In order to understand better the peculiar physical and roentgen changes produced by nonopaque foreign bodies, it is important that one have a basic knowledge of the respiratory mechanics of the bronchial tract. It must be remembered that the bronchi are not rigid tubes and that during inspiration they elongate and widen; conversely, on expiration they become shortened and narrowed. This constant changing of

the caliber of the bronchial lumen during respiration plays a major role in the production of the physical and roentgen abnormalities commonly found when foreign bodies invade the bronchial tract.

As a rule, once a nonopaque foreign body, particularly one vegetal in type, becomes lodged in a bronchus, a local reaction soon follows, producing severe edema of the bronchial mucosa, which further increases the bronchial obstruction. On inspiration, the bronchi being normally widened, the invasion of a bronchus by a foreign body at first produces only partial obstruction during this phase of respiration. Sufficient space still remains between the foreign body and the bronchial wall to allow the ingress of air. However, on expiration the situation becomes entirely reversed. The bronchi being now normally narrowed, the foreign body and the resulting local mucosal edema will in a varying degree obstruct the egress of air from the portion of the lung involved. This state of expiratory obstruction results in hyperdistention

Fig. 4.—Photograph of the foreign body, the rubber sac of a fountain pen, removed from the left main stem bronchus, where it had been lodged for a period of nine weeks. It measured 1.5 cm. in length and 1 cm. in width. When the object was viewed through the bronchoscope, the open end of the sac presented.

of the pulmonary segment immediately distal to the obstructed bronchus. In other words, a condition of obstructive emphysema is in the making. The mechanics involved in producing this condition is often referred to as an expiratory check valve mechanism. If there is any delay in the removal of the foreign body during this stage of obstructive pulmonary emphysema, the increased mucosal swelling and secretion resulting from the prolonged local irritating action of the foreign body will then completely cork the bronchus, thereby preventing air from entering or leaving the portion of the lung supplied by the obstructed bronchus. The mechanism, which previously was only expiratory in type, has now changed to that of a complete check valve obstruction, and the state of the lung, formerly emphysematous, is now completely atelectatic.

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INDUCTION OF ANESTHESIA WITH THIOPENTAL SODIUM IN TONSILLECTOMY

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THIS paper is a short note on my experiences with the intravenous administration of thiopental sodium (pentothal sodium®) in tonsillectomy in the past two years.

Intravenous anesthesia was rightly adopted only with considerable caution by members of the medical profession, as on theoretic grounds there were serious objections at first. There was a very real danger of injecting too much of the drug and of being unable to reverse the process.. Some of the earlier derivatives of the barbiturates were much more slowly eliminated than those in use today. Thiopental sodium was introduced to the medical profession in 1934 and has the advantages of rapid action and relatively short duration. It is broken down rapidly, partly in the liver, and is excreted by the kidneys in an unidentified form. It is unlike the slower-acting barbiturates, a good deal of which may be excreted from the kidney unchanged over a period of two to three days.

Thiopental sodium has been in general use for over twelve years in a gradually increasing proportion. As an example, I quote from the surgical register of the Ottawa Civic Hospital for January 1947, which was an average month. There were 468 operations under thiopental sodium anesthesia and 206 under all other types of anesthesia—ether, spinal and local. By now, a wide experience has been gained of the characteristics of thiopental, and, on account of its relatively low toxicity and its rapid breakdown and elimination, it is generally regarded as a fairly safe anesthetic. Like all barbiturates, it is a respiratory depressant and is likely to depress the reflex mechanism of the carotid sinus and the aortic nerves. The respiratory center loses its sensitivity to carbon dioxide. When there is a high concentration of carbon dioxide in the blood with a low concentration of oxygen and a depressed center, there is considerable danger, and it is believed that sudden deaths have been due to this state (Adams [1]).

Read at the first meeting of the Canadian Otolaryngological Society at Minaki Lodge, Ontario, on June 23, 1947.

1. Adams, R. C.: Intravenous Anesthesia, New York, Paul B. Hoeber, Inc., 1944.

The manufacturers of thiopental sodium and its medical sponsors all stressed that its use was undesirable in operations on the nose and the throat because there was great danger if the air passages should become obstructed. The same objection, of course, applies to any other anesthetic. Adams advised against its use in operations on the throat on the ground that it is difficult to prevent secretions from obstructing the air passages and on account of the persistence of reflexes in these regions, for which an unwarranted depth of anesthesia must be maintained to keep the patient quiet.

For a number of years I had had a desire to try thiopental sodium in tonsillectomy in adults, since to my mind it offers some advantages over ether. However, on account of the explicit warnings regarding its dangers and the possible medicolegal implications in case of accidents, it was not until 1945 that I began to use it systematically.

Hutchison, Mitchell and McHugh [2] published a paper in 1938 giving their experiences with intravenous anesthesia in a series of 22 children with tuberculosis and other pulmonary conditions in which ether was considered to be contraindicated. The average age was 8.9 years, the youngest child being 3 and the oldest 16. Five of the patients had thiopental sodium and the rest hexobarbital soluble (evipal soluble®). Hutchison and associates preferred hexobarbital soluble as being less depressing. Slight cyanosis was noted in some cases.

I have had no personal experience with the use of thiopental sodium in children except in 1 case, about ten years ago. I was about to do a bronchoscopy for a foreign body and discussed the question of the anesthetic with the anesthetist. As ether vapor obscures the field, thiopental sodium was decided on. The patient stopped breathing almost at once, and, in spite of the immediate introduction of a bronchoscope and continued artificial respiration, he did not recover. We believe that too large a dose was given too rapidly. Since that time, I naturally have been reluctant to use thiopental sodium in children.

During the war it happened that I had large numbers of tonsillectomies to perform on service personnel, and, like most others in a similar situation, I had considerable difficulty with inducing anesthesia by means of ether, even when administered by experts. Men under training seemed to acquire a high resistance to ether, whether given by the open method, by pharyngeal tubes or intratracheally. Fairly heavy premedication with pentobarbital sodium was necessary, and never less than ¼ grain (15 mg.) of morphine sulfate was needed to keep the patients reasonably quiet. Even so, it was often necessary to give chloroform as well to abolish straining and violent coughing. This

2. Hutchison, K.; Mitchell, H. S., and McHugh, H.: Canad. M. A. J. **39**:237 (Sept.) 1938.

straining produced increased bleeding, necessitating the excessive use of suction, which, of course, drew away the vapor of the anesthetic, making the anesthesia lighter than ever and producing more straining. This method was thoroughly unsatisfactory, and at last, with the cooperation of Dr. H. T. Jost, the chief anesthetist, it was decided to try thiopental sodium.

At first we gave straight thiopental sodium and found that one pleasing feature was the immediate relaxation of the jaw. Breathing was quiet and regular within a minute or two, and the operation could be started within five minutes, instead of the usual twenty to twenty-five minutes. If we waited a few minutes after apparent complete anesthesia before beginning the operation, we had very little trouble with laryngeal spasm, but if one touched the throat before the patient was completely relaxed, sometimes severe laryngeal spasm occurred. After encountering a few of these spasms, we made it a rule to have a laryngoscope, an intubation tube and oxygen always at hand, though at that time we used them only occasionally. Once the patient had reached the proper level of anesthesia, the operation could be performed in comfort, with noticeably less bleeding than with ether. When spurting vessels were encountered, the suction tip could be used as much as desired until the ligatures were applied. The operation itself was much shortened, quite apart from the saving of time in the induction.

Another agreeable feature of use of thiopental sodium is the absence of nausea and vomiting during the period of recovery. After waking up, the patient usually dozes for several hours, which result is also desirable during the first painful period.

Not long after we had used this anesthetic in a few cases patients heard about it, and they began asking for thiopental sodium rather than ether. The number of cases on which this paper is based is rather small, just under 200. This fact is due to various causes, the first of which is the overcrowded state of hospitals and the low priority given to patients requiring tonsillectomy. After some of us had begun the use of thiopental anesthesia in tonsillectomy, many general practitioners, with little or no experience in the use of the anesthetic, began doing likewise and giving dangerously large doses, with the result that the hospital authorities became alarmed and prohibited the use of the drug for several months, while the whole question was reviewed and opinion sought from other hospitals. It is interesting to note that the adverse opinions received were all from physicians who had never used the drug and whose opinions therefore were of little value. Eventually the ban was lifted, but the administration of thiopental sodium in tonsillectomy was limited to that by staff anesthetists, which rule I consider a wise one. I would definitely state that thiopental sodium should not be admin-

istered in operations on the throat unless the anesthetist has had extensive experience in its general use and is capable of doing a rapid intubation.

After finding that thiopental sodium alone was fairly satisfactory in tonsillectomies, I carried out another series of cases, with pernasal intratracheal intubation and a steady flow of oxygen. This method was even more satisfactory, as we were certain of an adequate airway and supply of oxygen. The assistant, meanwhile, could depress the tongue without choking off respiration. I do not much like peroral intubation for tonsillectomy, as I find that the tube gets in my way, though I do not hesitate to use it if necessary. A tube running down from the back of the nose, on the other hand, is not in the way at all.

Adams advised the spraying of the pharynx and larynx with cocaine or other local anesthetic before introduction of the intubation tube. This has not been done in any of my cases and does not seem necessary if the proper level of anesthesia has been reached first. If necessary, a small amount of nitrous oxide or cyclopropane is given to produce sufficient relaxation of the larynx. In many of my cases the patient was carried along with thiopental sodium with the addition of oxygen delivered through double nasal tubes into the pharynx. This method works fairly well but requires more care to see that the tongue is kept forward.

DOSAGE

Doses varied from 0.75 to 1.25 Gm. in average cases. Exceptionally, 2 Gm. was necessary. A 5 per cent solution was generally used, though for some time one anesthetist used a 2.5 per cent solution. The amount of thiopental sodium varied considerably, according to the amount of premedication. Dr. D. W. Whyte,[3] in the Royal Canadian Air Force, gave 1½ grain (0.097 Gm.) of pentobarbital sodium the night before operation and again in the morning, one and one-half hours before operation, followed by ¼ grain (0.01 Gm.) of morphine sulfate and $\frac{1}{75}$ grain (0.8 mg.) of atropine for husky men or ⅙ grain (10 mg.) of morphine sulfate and $\frac{1}{100}$ grain (0.6 mg.) atropine for women and less robust men. He stated, "This rather heavy premedication markedly decreases the dosage of pentothal necessary during the operation."

There is room for a difference of opinion on this point. I am not convinced that it is an advantage to have the patient heavily drugged with substances which are slowly eliminated in order that less thiopental sodium, which is quickly detoxicated and eliminated, may be given. As a matter of fact, in my series we have got along satisfactorily with ¼ grain morphine sulfate administered half an hour before operation, without any pentobarbital sodium or other barbiturates.

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3. Whyte, D. W.: Anesth. & Analg. **25**:64 (March-April) 1946.

COMBINED ANESTHESIA

A combination of thiopental sodium with cyclopropane or nitrous oxide or ether is widely used in order to lessen the amount of thiopental sodium required. It is generally considered to be safer than thiopental sodium alone when given intratracheally. If necessary, the anesthetic vapor can be instantly switched off and oxygen substituted. I might mention that there is a tendency for the patient's color to be a little duskier than with ether, and we now make it a rule to have oxygen delivered into either the pharynx or the trachea during the operation. I have performed a fair number of operations with cyclopropane and thiopental sodium but it is difficult to decide whether it is better to give thiopental sodium with or without cyclopropane. Both methods were completely satisfactory. The combined administration does facilitate intubation, but after that point it seems to be unnecessary. In some cases we used thiopental sodium and ether, and, as far as the operation was concerned, the combination was satisfactory, but the patients had some of the usual after-effects of ether, though to a lesser degree.

Curare is a useful adjuvant to thiopental sodium in securing easier intubation. As is well known, curare paralyzes the motor nerve endings. The muscles are affected in the following order: first, those muscles supplied by the cranial nerves; next, those of the trunk and extremities, and then the muscles of respiration, with the diaphragm last of all. Recovery occurs in the reverse order. A dose of 60 to 70 mg. is given intravenously at the beginning of the anesthesia and takes effect within one minute.[4]

Nikethamide was given at the end of the operation in some of our earlier cases to hasten the return to consciousness. This practice was usual among the anesthetists at that time with other types of operations. In others caffeine was given instead, but it was difficult to know how much effect these drugs had, as the patients were about to wake up in any case. In our later cases, neither nikethamide nor caffeine has been given, and the patients seem to wake up just as quickly, so that I should say that in a short operation, such as tonsillectomy, they are unnecessary.

COMPLICATIONS

The most serious difficulty encountered was laryngeal spasm. This can be quite alarming, and the patient may become deeply cyanosed. As mentioned elsewhere, it is usually due to stimulation of the throat before the anesthesia is deep enough. As the depth of anesthesia cannot always be determined with certainty beforehand, it is a good idea for one to give cyclopropane for a minute or two before introducing the tracheal tube.

4. Cole, F.: Anesthesiology **6**:48 (Jan.) 1945.

Once the tube is in place, administration of the cyclopropane can be discontinued and oxygen substituted. If a severe spasm has occurred, a tube must be put in at once to supply oxygen and relieve the spasm. It is not safe for one to rely on giving more thiopental sodium.

Occasionally patients become excited and uncontrollable on being sent back to the ward. This occurrence is rare and may perhaps be due to some difference in premedication. It has not happened with more than a few in this series. Most had a peaceful recovery.

In the early days of intravenous therapy there were some sore arms from the extravenous injection of the anesthetic fluid, but they are happily rare now, as the anesthetists are more expert.

SUMMARY

Thiopental sodium in the hands of an experienced anesthetist is a satisfactory anesthetic for tonsillectomy, but it is not safe in inexpert hands.

With intratracheal insufflation of oxygen, the dangers of anoxia and toxic effects are avoided.

Induction and recovery are rapid.

The tendency to hemorrhage is somewhat less than that with ether.

Absence of nausea and vomiting is an advantage.

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SOLITARY MYELOMA MISTAKEN FOR FRONTAL SINUSITIS

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IN FULMINANT frontal sinusitis, besides characteristic frontal pain, intranasal obstruction and purulent discharge, swelling of the periorbital tissues and protrusion of the globe frequently are seen. If these objective evidences are presented in a case with a history consistent with acute sinusitis, one might be excused for mistaking this common condition for the much rarer occurrence of myeloma involving the accessory nasal sinuses. Such a case is reported here.

REPORT OF A CASE

F. B., a white man aged 29, was a pipe fitter and welder and had served in the Navy in World War II. He was first seen by me on Jan. 25, 1947 with the complaint of "sinus trouble" for nine weeks. He had a headache of the left frontal area; proptosis of the left eye, which protruded outward and downward; nasal stuffiness, and dark reddish discharge from the left naris.

History.—The patient had frequented commercial swimming pools a great deal during the summer of 1946. In September 1946, he had been in bed for ten days with a "head cold," at which time he had had a temperature of 100 F., nasal blocking and watery discharge (no pus). There had been, he thought, some swelling about the left eye at that time. One month after the cold there had been a bilateral frontal headache, brief pains in the nape "four or five times a day" and a dark blood-stained discharge from the left naris, but no nasal obstruction. The patient's physician had sent him to a roentgenologist, who diagnosed the condition as "sinusitis" and gave the patient weekly roentgen treatments. His physician gave him injections of penicillin daily during this time. The roentgenologic report was "clouding of the left antrum and frontal sinus, with decalcification of the floor of the frontal sinus."

Examination showed protrusion of the left eye and tenderness over the floor of the left frontal sinus and the front of the left maxillary sinus. The left middle meatus exhibited "hypertrophy" and contained a little dark bloody discharge. Transillumination revealed involvement of the sinuses as follows: right frontal sinus, 2 plus; left frontal sinus, 4 plus; right maxillary sinus, 2 plus, and left maxillary sinus, 4 plus. On attempting lavage of the maxillary sinuses, I found it impossible to wash through the left. Posterior rhinoscopy showed dark red material in both the middle and the superior meatus on the left side. The ocular abnormality on the left side was as before mentioned. Examination of the blood on the patient's entering the hospital disclosed no abnormality.

There were no complaints directed toward the rest of the body, and examination did not indicate any metastases at that time. The condition was therefore considered "solitary."

My conception of the case was sinusitis, in which there was not the usual purulent discharge because of intensive medication and roentgen therapy. I stated the belief that the floor of the left frontal sinus and the lamina papyracea had probably been destroyed by the intense involvement, the destruction accounting for the ocular protrusion. The thought of a malignant growth, of which I have seen several in my private practice, and which causes the bloody character of the sinal production, was countered by my not finding the wall of the maxillary sinus softened, like pasteboard, when I introduced the antrum needle.

The patient was given penicillin and sulfadiazine on entering the hospital; a 1 per cent solution of phenylephrine hydrochloride (neo-synephrine hydrochloride●) was used intranasally, and the left frontal region was treated by exposure to infra-red radiation. Urinalysis, determination of hemoglobin and a cell count afforded no diagnostic data.

No improvement having occurred after hospitalization and treatment, on January 27 I did the Boies external drainage operation on the left frontal sinus, cleared out somewhat the middle meatus on that side and performed the Halle window operation on the left maxillary sinus. No pus was found in any of these sinuses of the anterior series but only serohemorrhagic fluid in the frontal sinus and excess bleeding from the maxillary sinus and the middle meatal tissues. Culture of the fluid from the frontal sinus produced nonhemolytic streptococci. No decrease in the prominence of the left eye followed the operation, though the medication and drainage given this patient were expected to produce prompt improvement in the inflammatory involvement of the frontal sinus. However, pain in the left frontal region was not complained of after the operation. It soon became evident that the proptosis was actually increasing, and then edematous swelling over both frontal regions was noted. This situation suggested that osteomyelitis was developing. The roentgenologist's findings were in agreement with this conception, his report reading as follows: "Over the left frontal sinus there is an area of rarefaction, with a suspicious area, suggesting destruction, in the posterior wall of the sinus."

I then felt that removal of the frontal bone would be necessary, and therefore, on February 17, the second operation was performed. With the use of local anesthesia, an incision was made across the frontal regions, passing below the brows, dipping down to the glabella at the center and curving upward at the outer ends, so as to permit ready reflection of the soft parts upward from the frontal bone. Necrosis of the margin of the right superciliary ridge was found, and the surface of the right frontal bone had a slightly peppery appearance, such as is seen in a mastoid bone the surface of which is barely affected. The periosteum over the involved portion of the right superciliary ridge was found separated from the bone. When the frontal sinuses were opened, thick, grayish brown, syrupy material oozed out of the right one, without pulsation. There was a large fault in the posterior walls of both frontal sinuses; the floors were largely destroyed, and no septum was found. The lining of these sinuses was thick and bled freely; bared areas of the walls were rough and hard. Just to the left of the midfrontal region was a thick ridge, and just to the left of that ridge the bone was somewhat thin—in the area which I had supposed to be softened by the suspected "osteomyelitis." There was no softening of the bone, such as is found in osteomyelitis. On removal of the rest of the posterior walls of the frontal sinuses, pus escaped from the epidural region on the right, and the dura there was observed to be thick and shaggy. In these sinuses no pus was found, but only a thickened, bleeding membrane. In fact, this excess bleeding made it impossible for me to do thorough cleaning up of the frontal sinus or the exenteration of the ethmoid cells. A branch of the superior longitudinal sinus bled freely, making it impossible to remove as much of the

frontal bone as I had intended doing, in accordance with the procedure taught by the Mosher school for handling osteomyelitis of the frontal bone. The cavity was sprinkled with sulfathiazole powder and packed with iodoform gauze and the incision loosely closed with several interrupted sutures of black silk, leaving an opening at the center for the end of the iodoform gauze drain. A specimen was sent to the laboratory for microscopic examination.

The laboratory report was "plasma cell myeloma." After receiving it, I began to reread all available recent publications on multiple and solitary myeloma.

Bayrd and Heck, of the Mayo Clinic, in January 1947 published a thorough and informative review [1] of the subject. Their paper, however, offered little encouragement as to treatment and prognosis. I was particularly interested in a paper by Figi, Broders and Havens,[2] published in June 1945, in which they reported 11 cases of solitary plasma cell myeloma of the upper respiratory tract. The authors stated that Claiborn and Ferris had found only 12 cases involving the nose and pharynx prior to 1931, that Cappell had found only 2 cases in 122 cases of tumors of the nose and throat, that Mattic and Thibaudeau had found 19 cases of plasma cell tumor of the upper respiratory passage reported in the literature prior to 1935, in addition to 1 which they had observed, and that Jackson, Parker and Bethea had studied 17 cases of multiple myeloma, in 5 of which solitary plasma cell myelomas had preceded the generalized involvement.

That plasma cell tumor is rare is attested by the fact that of 626 cases of malignant growths in the upper respiratory tract, this tumor was found in only 6.

Treatment.—When the pathologist's diagnosis of "plasma cell myeloma" was announced, on March 7, radium and roentgen radiation was discussed with the roentgenologist, and the article by Figi and associates was called to his attention, since I was enthusiastic about the results those men had obtained at the Mayo Clinic. Contrary to my inclination, the head of the roentgenologic laboratory did not use radium but depended on roentgen therapy.

The use of "stilbamidine" (4, 4-stilbenedicarboxamidine), as reported by Snapper [3] in January 1947, gave my associates and me some encouragement, and Merck & Co. furnished this material for our use. The first dose administered was 50 mg.; the second, 100 mg. two days later, and those thereafter, at two day intervals, 150 mg. in 10 cc. of distilled water. The total amount of "stilbamidine" given was 3,000 mg., from March 29 to May 8; one more dose of 150 mg. was given on June 12, but an unpleasant reaction discouraged its further use.

It may be of some help to others who use "stilbamidine" for me to describe the patient's reaction to the drug: After the intravenous injections, though they were given very slowly and carefully, the patient experienced flushing and a tingling and burning sensation, with profuse perspiration.

Strong sedatives were necessary for pain in the left shoulder and arm. Motion in this joint became more and more restricted. Otherwise, however, the patient seemed remarkable for his noncomplaining attitude.

On March 11 the patient was unable to void. The urologist (Dr. Joseph F. Corriere) was consulted, and an indwelling catheter was inserted and irrigations

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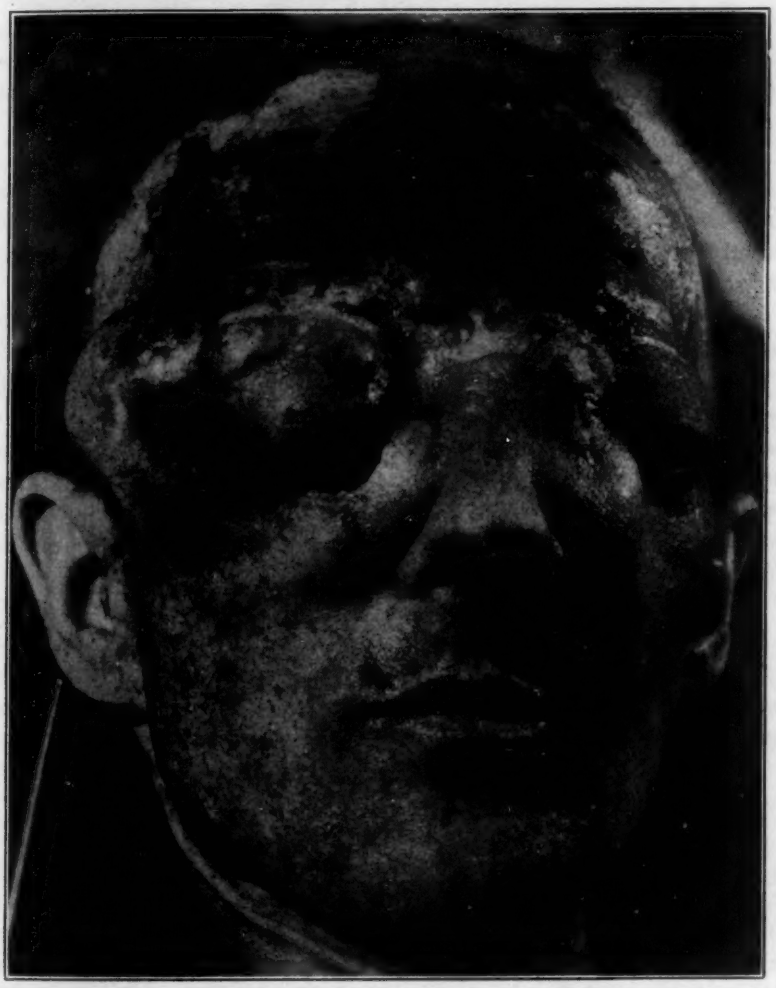
1. Bayrd, E. D., and Heck, F. J.: Multiple Myeloma, J. A. M. A. **133**:147-157 (Jan. 18) 1947.

2. Figi, F. A.; Broders, A. C., and Havens, F. Z.: Plasma Cell Tumors of the Upper Part of the Respiratory Tract, Ann. Otol., Rhin. & Laryng. **54**:283-297 (June) 1945.

3. Snapper, I.: Stilbamidine and Pentamidine in Multiple Myeloma, J. A. M. A. **133**: 157-161 (Jan. 18) 1947.

were kept up. The original swelling around the left orbit was greatly augmented by enlargement of that region, the disfigurement ultimately coming to be that shown in the accompanying photograph. Swelling of the right eyelids occurred late in the disease, and the right globe was pushed downward somewhat. On May 9 the patient complained of feeling "numb from the waist down." Later he became unable to move the lower extremities.



Photograph of the patient in the late stage of the disease, showing the disfigurement after the original swelling around the left orbit had been greatly augmented by enlargement of that region.

The roentgen treatments did not halt the enlargement of head structures, nor did they give the patient any relief. The "stilbamidine" seemed equally unhelpful, and the patient felt, finally, that it caused nausea. The patient died of bronchopneumonia on June 20.

The findings at autopsy did not correspond with the clinical manifestations, such as the paralysis of the lower extremities, the paralysis of the bladder, the painful and immovable shoulder joint and the vacuolation found in the roentgenograms of the humeral head and the pelvic bone. (The exposure of the spinal cord was carried only to the eighth thoracic vertebra.)

SUMMARY

Plasma cell tumors, plasmacytoma and multiple myeloma are one and the same disease, and I think it desirable that writers mention all these terms in describing a case, until this rare condition becomes more generally understood by the medical profession.

Multiple myeloma applies to the condition when it has become disseminated, the original site not having been recognized while the tumor was solitary. Reports of solitary myeloma have been made recently, especially by Figi, Broders and Havens. In my case the lesion was solitary when first seen.

Important is the now well established fact that these tumors do not have to be considered of bone marrow origin, as was originally believed.

Solitary myeloma is most frequently found in the pharynx, palate, nose and accessory nasal sinuses, but it may occur "behind the ear, in the mastoid cells, on the alveolar process, on the scalp, on the conjunctiva, on the arms and on the tongue."[2] Figi, Broders and Havens reported 11 cases in which the areas involved were as follows: the maxillary sinus, 6 cases; the oropharynx, 2 cases; the nasopharynx, 2 cases, and 1 case in the larynx.

Early biopsy of atypical growths in the nose and throat is especially suggested after a review of this case. The softened walls of the maxillary sinus in carcinomatous involvement do not occur in plasma cell growths, but there are rapid appearance of abnormal tissue, bleeding and disfigurement.

"Stilbamidine" was not of benefit in this case.

Bence Jones proteinuria did not occur in this case.

The diagnosis was confirmed by the National Pathological Institute, to which a slide was sent by our laboratory.

Since this patient, and 1 other I have subsequently known of, exhibited an unusual absence of complaining (save for localized joint pain), it will be interesting if subsequent reports of cases will note whether or not euphoria is one of the characteristics of this disease.

230 East Broad Street.

Case Reports

PERFORATING BURN OF THE TYMPANIC MEMBRANE

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ACCIDENTAL burn of the tympanic membrane is uncommon. Recent bibliographic summaries do not mention it. Beck described a case in which the tympanic membrane had been destroyed by pure phenol which was instilled into the ear by a soldier in an attempt to avoid front line service. Recovery was good.[1] It is difficult to visualize a hot liquid accidentally splashing directly into the external auditory canal and burning it and the tympanic membrane without involving the pinna or some other facial area. One thinks of a factitial lesion when such an injury is seen. However, after detailed questioning of my patient I am convinced of the truthfulness of his story. Although his injury came under the Workmen's Compensation Code of the State of New York, his interest was not in any monetary remuneration. He was the father of 6 children and realized that whatever compensation he might receive for a moderate unilateral impairment of hearing would contribute little to the support of his family. He further realized that defective hearing in one ear might hinder him considerably if and when he found it necessary or desirable to change jobs. He was interested in securing a return of normal hearing and alleviation of his symptoms.

REVIEW OF A CASE

G. E. C., a 31 year old white man employed as an automobile mechanic, was first seen on May 22, 1947. Five nights previously he was underneath a truck that had just completed a run, getting ready to drain the crankcase oil. As he removed the motor pan oil plug, a hot liquid from somewhere off the motor block dripped into his right ear. He thought it was gasoline because of the immediate taste. He suffered immediate excruciating pain and in fifteen to twenty minutes consulted a physician. The latter instilled several drops of a 4 per cent cocaine hydrochloride solution after syringing the ear with "warm water." Pain persisted. The night of the accident and the next day there was a slight but steady bloody discharge from the ear. The patient again consulted his physician, who referred him to me on May 22.

The patient complained of intermittent severe temporal pain, tinnitus, defective hearing and bloody discharge from the ear—all on the right side. He stated that he had not noted vertigo or previous aural disease.

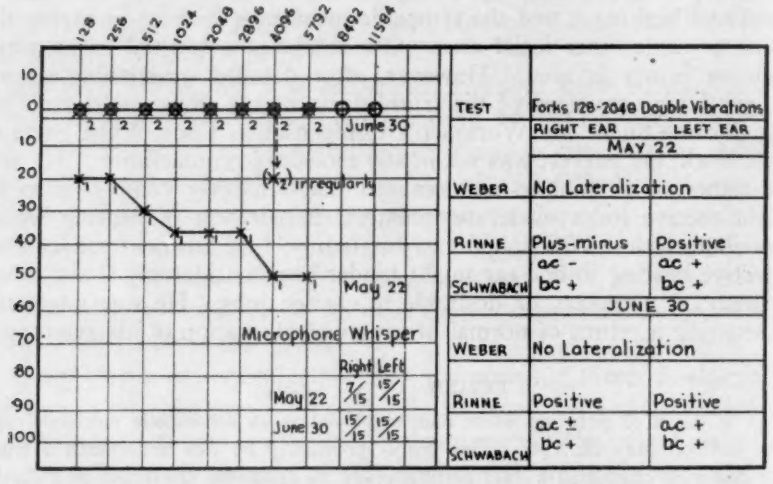
The left ear was normal. There was no evidence of a burn on the face or the pinna. No spontaneous nystagmus on lateral gaze was noticed. The right external meatus and canal were red and edematous, and the epithelium was sloughing. The tympanic membrane was diffusely hyperemic and edematous. Adjacent and

1. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1930, p. 413.

posterior to the umbo was a 2 mm. oval perforation. There was no discharge from, or pulsation in, the middle ear. What mucosa could be seen was fiery red. On the posterosuperior quadrant of the tympanic membrane there was a denuded area covered with flecks of old blood, suggestive of a ruptured bleb. Flecks of fresh blood were scattered over the membrane. Audiometric study carried out in a room in which noise was at 10 decibels showed a 26 per cent impairment of hearing for air-conducted sounds in the right ear, with the Rinne test giving a plus-minus result, the Weber test sound not lateralized, and the Schwabach test showing air and bone conduction reduced.

My diagnosis was second and third degree burns of the external auditory canal and tympanic membrane, with perforation of the tympanic membrane and moderate secondary impairment of air conduction hearing, probably due to a chemothermal agent.

The ear was cleansed with sterile dry wipes, and a sedative drug was prescribed. The patient was instructed to let "nothing whatsoever" enter the ear



Audiogram of a patient with a perforating burn of the right tympanic membrane. O's represent the threshold of hearing of the left ear; X's, that of the right ear.

and was kept under daily observation and "careful neglect." On May 26 the perforation of the tympanic membrane had sealed over. The posterior half of the membrane remained beefy looking, and there was some retraction of the anteroinferior quadrant. Tinnitus and defective hearing persisted. Pain had disappeared.

No remarkable change was noted until June 5, when there was a recurrence of pain with bulging of the posterior half of the tympanic membrane. There was no history of recent infection of the upper respiratory tract. Myringotomy, performed after induction of local anesthesia,[2] revealed pus under pressure. The patient was treated with 300,000 units of penicillin in oil and wax, also with sulfadiazine 30 grains (1.94 Gm.) initially, and then 15 grains (0.97 Gm.) every four hours, and with a 1 per cent solution of "tuamine sulfate" (2-amino-

2. Equal parts of cocaine, phenol and menthol crystals were applied, and neutralized calcolid.

heptane sulfate). The myringotomy wound showed a slight purulent discharge until June 9, after which there was no evidence of active suppuration. The retraction of the anterior portion of the tympanic membrane was more definite. Moderate hyperemia and edema of the posterior portion persisted.

On June 14 catheteral insufflations of the eustachian tube were begun simultaneously with applications of 8 pounds of negative pressure to the external auditory canal. After two treatments, administered at daily intervals, there was a noticeable improvement in the appearance of the drum. On June 30 all landmarks of the tympanic membrane were normal. Audiometric study showed no gross loss of hearing. The Rinne test showed air conduction better than bone conduction. In the Weber test, sound was not lateralized, and slight reduction of hearing by air conduction was shown by the Schwabach test.

Reviewing this case, I feel that I should have begun antibiotic and sulfonamide therapy immediately, even though, when I first saw the patient, there was no local or systemic evidence of gross suppuration. Here was an exposed middle ear behind a severely burned external auditory canal and thermally perforated tympanic membrane that had been subjected to syringing with "warm water" and an instillation of drops. Perhaps if I had, the complicating suppurative otitis media could have been prevented.

Abstracts from Current Literature

Ear

THE REPAIR OF THE EAR DRUM IN BLAST INJURIES. SHIRLEY HAROLD BARON, Ann. Otol., Rhin. & Laryng. **57**:143 (March) 1948.

Rupture of the ear drum was the commonest early aural lesion encountered in cases of blast injuries in World War II and has been the commonest in peacetime. Air-borne pressures are considerably less than water-borne ones. The critical distance from the center of blasts in air is said to be 20 feet (6 meters), that in water, about 80 feet (24 meters). There are two phases of the atmospheric blast: a powerful and shorter pressure component and a weaker and longer suction component. There are also two phases of immersion blast: the "a" phase, or compression wave, which is the destructive factor, and the "b" phase, which is the disturbance caused by the expanding bubble of gases liberated by the explosion; this phase is not destructive.

Damage to the ear drum ranges from small interstitial hemorrhage to almost total destruction. It is agreed that rupture of the drum seems to exert a protective influence on the labyrinth and cochlea and that the degree of deafness is greater when the drum is not ruptured; this has been confirmed experimentally. Deafness is of the middle ear type superimposed on a temporary perception deafness. Permanent loss of hearing is usually not pronounced. Tinnitus is invariably present but generally disappears. In perforations occurring in peacetime, the injury has no definite location, but the majority of those due to war blasts are in the antero-inferior or central quadrant. Perforations of the attic are rarely seen; some say they do not occur. Even the extremely large perforations may close without leaving a scar. Even in cases with infection, the drum tends to close after cessation of the discharge. The opinion exists that the perforation tends to heal better if there has been infection and that the reaction of inflammation is a stimulus to repair. Continued infection hinders the closure of the perforation, but after cessation of discharge closure may be aided by stimulation of the edges of the perforation and use of a splint. The latter should not not be used in the presence of moisture from the middle ear. Cargile membrane, cigaret paper, thin layers of oiled cotton, rubber tissue, cellophane, fibrin film and gold foil have been used; the first, probably the most useful, is applied to the perforation (one fourth to one half larger than the opening) one or two days after the epithelium along the margins has been scarified or cauterized with trichloroacetic acid.

MILLER, Philadelphia.

THE EFFECTS OF NEGATIVE AIR PRESSURE IN THE MIDDLE EAR. E. G. WEVER, MERLE LAWRENCE AND K. R. SMITH, Ann. Otol., Rhin. & Laryng. **57**:418 (June) 1948.

The authors carried out their experiments on cats, making use of the electrical potential of the cochlea to show how the ear's efficiency in sound reception is affected by pressure changes. Previous studies had been made on the effects of positive pressure in the middle ear. The effects of the negative pres-

sure closely approximate those of positive pressure. The sensitivity is reduced, but the form of the intensity function over the greater part of its course is essentially unaltered. Effects occur for both air-conducted and bone-conducted sounds, but their amounts differ greatly. There is a curious instability in an ear exposed to changes in pressure. After any change of pressure a period of slow adjustment usually occurs, during which the potentials settle down only slowly to an equilibrium value. Typically, when a positive or negative pressure is introduced, the response falls abruptly at first, rises rapidly for a time and then makes a slow approach to a final value. In some instances the rise overshoots the final value and settles down to it, or even crosses it waveringly several times before settling down. The amount and rate of this process of equilibration vary greatly with different animals; often it takes as long as two minutes. It is certain that this instability is not an activity of the muscles of the middle ear, for it occurs in curarized animals and is seen even after death. The evidence indicates that the pressure operates mainly on the transmission mechanism, especially the ear drum, and its action is to increase both the stiffness and the damping of these parts. Some effect also is referred to the inner ear.

MILLER, Philadelphia.

ELECTROCOAGULATION OF THE MEMBRANOUS LABYRINTH. LEROY A. SCHALL AND J. H. TOM RAMBO, Ann. Otol., Rhin. & Laryng. **57**:590 (Sept.) 1948.

Schall and Rambo carried out experiments on monkeys to determine whether the Day procedure for electrocoagulation of the membranous labyrinth could be so modified as to improve the chances for retention of hearing. Seven ears in 5 monkeys were operated on with Day's technic, and the monkeys then killed, from ten to forty days after operation. Histologic examination of the sections showed no significant pattern as to the amount of damage done to the cochlea, either in relation to the time elapsed after the operation or to the amount of coagulating current used. The authors concluded that the damage to the cochlea is unpredictable and that retention of serviceable hearing after the operation is improbable. On the other hand, the elimination of vertigo in Ménière's disease may definitely be expected, for when the optimum current is used the damage to the membranous labyrinth of the vestibule and semicircular canals is complete and predictable. No damage to the facial nerve was noted in any of the ears.

MILLER, Philadelphia.

GROUP PURE TONE HEARING TESTING IN THE PUBLIC SCHOOLS. HAYES A. NEWBY, J. Speech Disorders **12**:357 (Dec.) 1947.

A new group pure tone hearing test was administered to 1,200 subjects from the twelfth through the second grade. Experimental testing was conducted with samples of the population from the eighth through the second grade to study the reliability and validity of this test and its efficiency as a screening device. The results were as follows: 1. Ninety per cent of the ears showed differences between the results of the group test and the results of the group retest at the frequencies tested of 5 decibels or less. 2. Of the entire group of 188 ears in the validity studies from the eighth through the third grade, almost 90 per cent showed differences between the results of the group test and the results of individual tests at the frequencies tested of 10 decibels or less. 3. The group pure tone test operates most efficiently as a screening device when a significant hearing loss on the group test is defined as one of 20 decibels or more for any frequency for either ear. 4. Use of

the 20 decibel loss as the significant criterion results in a correct classification of 80 per cent of the ears, the needless retesting of 18.6 per cent of the ears and the failure to discover significant losses in 1.6 per cent of the ears. 5. On the basis of this study, the group pure tone test appears to be superior to the group phonograph speech audiometer as an efficient screening device.

PALMER, Wichita, Kan.

POSTOPERATIVE PROBLEMS IN CASES OF FENESTRATION. M. C. HICGUET and M. VAN EYCK, Rev. de laryng. **69**:301 (July-Aug.) 1948.

The authors report observations in 48 cases; they noted the appearance of a homolateral nystagmus during the burring of the semicircular canal, the nystagmus becoming contralateral as soon as the perilymphatic space was open and subsiding in two or three days. The response was probably due to rupture of the pressure balance between the perilymph and the endolymph, and not to postoperative serous labyrinthitis. The latter manifests itself only by a decrease in auditory gain. To prevent this secondary labyrinthitis, the authors use intravenous injections of 10 cc. of isotonic solution of sodium chloride twice daily for three days and also inject 1 per cent procaine hydrochloride without epinephrine into the superior sympathetic cervical ganglion. This ganglion is known to send autonomic fibers to the labyrinth (Lériche).

The authors noted often improvement of hearing in the opposite ear after fenestration, the gain sometimes reaching 15 decibels. They attribute this change to bilateral reflex action of the sound waves on the tensor tympani muscles and to the crossed auditory pathways (found by Bremer in the cat). The auditory stimulus of the treated (good) ear would stimulate the sound ear.

They also found that the fistula sign subsides in most cases, as a result of the large flap covering the fenestra and the large size of the fenestra, the adhesions of the membranous labyrinth to the cutaneous flap forming a large thin scar.

HERSON, Chicago.

VASCULAR DISTURBANCES OF THE INTERNAL EAR (COCHLEAR BRANCH). J. S. RIESCO MACCLURE, Rev. de otorrinolaring. **8**:3 (March) 1948.

Cochlear disturbances occurring in persons of middle and old age are frequently attributed to vascular alterations. The author made a clinical investigation of the relation between these disturbances of the ear and vascular changes observed in such patients. He presents a series of 51 patients with cochlear disturbances (tinnitus and hypacusis) in each of whom the middle ear and eustachian tubes were apparently normal and in whom there was no evident cause for symptoms of inflammation, infection, trauma or otosclerosis. The age groups of these patients ranged from 50 to 70. Thirteen of these patients presented impaired hearing without tinnitus; 1 had tinnitus without impaired hearing and 37 had impaired hearing with tinnitus. Riesco MacClure believes that tinnitus is always accompanied with impaired hearing, that senile hypacusis may occur without tinnitus and that bilateral hypacusis may be associated with tinnitus which may be bilateral or localized in the ear most seriously affected or in the head.

He noted that the audiometric curve can indicate the extent of degeneration and atrophy of the basilar membrane in the organ of Corti. This relation is reflected by the patient's ability to hear the human voice. For instance, when the degen-

erative process involves the basilar turn, the upper tones are involved and the spoken voice can be heard at a distance of 5 meters. If, on the other hand, the atrophy extends into the second turn of the cochlea, which responds to impulses from 512 to 2048 double vibrations per second, the patient hears the spoken voice only with great difficulty at 5 meters. Finally, when the degenerative process extends to the apical turn of the cochlea, the patient has difficulty in understanding words at a distance of 40 cm.

The author analyzes a group of 49 patients on the basis of cochlear disturbances with and without vascular changes. Of these, 20 did not have any appreciable vascular change; in 17, the vascular changes were minimal and in the last group, of 12, there were advanced vascular changes, for which the patients were under constant medical treatment. Riesco MacClure concludes that there is neither clinical nor histopathologic differentiation of senile hypacusis, progressive hypacusis of unknown origin in young persons and hypacusis due to degenerative, atrophic neuritis in the cochlea. He believes the cochlear alterations that occur with old age are not due to vascular changes but, rather, result from a degenerative and atrophic neuritis.

PERSKY, Philadelphia.

Pharynx

ABERRANT SALIVARY GLAND TISSUE IN TONSILLAR FOSSA. J. J. O'NEIL, Eye, Ear, Nose & Throat Monthly, **27**:234 (May) 1948.

A review of 6 cases of aberrant salivary gland in the tonsillar fossa previously reported in the literature is presented. The author reports on what is apparently the seventh such case, which was of a 4 year old child on whom a tonsillectomy and adenoidectomy were performed. After removal of the right tonsil, a small, white mass of tissue was observed at the base of the right tonsillar fossa. This tissue was removed with a snare, and pathologic examination revealed it to be an essentially normal salivary gland. The postoperative course was uneventful.

JENNES, Waterbury, Conn.

ADENOIDS, THEIR VISUALIZATION AND REMOVAL. MERVIN C. MYERSON, Laryngoscope **58**:542 (June) 1948.

Myerson touches on some historic facts concerning adenoid tissue and methods for its removal and emphasizes the importance of thorough and complete removal. Direct visualization of the nasopharynx during and after the operation is recommended. The author uses the Goodwillie palate retractor as an aid in accomplishing this.

HITSCHLER, Philadelphia.

Larynx

LARYNGEAL EDEMA IN EPIDEMIC PAROTITIS. JAMES S. WALKER AND E. LAMONTE GANN, Ann. Otol., Rhin. & Larnyg. **57**:163 (March) 1948.

Edema of the larynx occurring with epidemic parotitis is a rare, but potentially dangerous, complication. The authors report 2 cases, apparently the first reported in America, although a few cases have been reported abroad. One of 2 patients also had presternal edema. Both complications, the cause of which is obscure, are probably due to obstruction to normal lymphatic drainage; this may be secondary to the pressure of the swelling. Mumps should be considered as a possible diagnosis in all cases of unexplained laryngeal edema, particularly

when associated with enlargement of the salivary glands. The diagnosis in isolated cases of epidemic parotitis is not always easy, especially in the adult, and a careful history of the patient is important.

MILLER, Philadelphia.

TREATMENT OF CARCINOMA OF THE PHARYNX AND LARYNX. J. I. KEMLER, Eye, Ear, Nose & Throat Monthly **27**:119 (March) 1948.

Fifty cases of carcinoma of the larynx and pharynx are presented. The author believes surgical measures are always indicated when carcinoma occurs in this region. He utilizes laryngofissure for intrinsic carcinoma of the larynx without fixation of the cords and laryngectomy when the tumor is more extensive or situated in the posterior third of the larynx, with fixation of the cord. Only in cases of extrinsic, far advanced carcinoma is irradiation considered beneficial, and then only as an adjunct following operation.

JENNES, Waterbury, Conn.

CARCINOMA OF THE LARYNX. LOUIS H. CLERF, F. JOHNSON BUTNEY AND JOHN J. O'KEEFE, Laryngoscope **58**:632 (July) 1948.

A masterful review and summary of cancer of the larynx is presented by Clerf, Putney and O'Keefe. It is clear and concise; almost every aspect of the condition is considered. The article is excellent for any one interested in the subject.

HITSCHLER, Philadelphia.

ACUTE INJURIES TO THE NECK INVOLVING THE FOOD AND AIR PASSAGES. M. LICHTENSTEIN, Surg., Gynec. & Obst. **85**:734 (Dec.) 1947.

The author reviews the case histories of 9 soldiers who sustained injuries to the neck involving the air and food passages and who were treated in an evacuation hospital during the Italian campaign in World War II. This particular type of wound was not common. The need for trachectomy was often present, but in many cases of wounds of the neck in which dyspnea was evident it was noted that incision into the fascial spaces, as part of the initial operation, was followed by relief from the signs and symptoms of obstruction.

Principal emphasis is directed to the problem of contamination of the deep structures of the neck from perforation of the oral, pharyngeal or esophageal mucous membranes. Incision into the fascial layers with exposure of the retropharyngeal space is advocated for the prevention of edema and the spread of infection into the mediastinum.

Attention is called to the value of gastrostomy for certain wounds of the pharynx and esophagus and the desirability of performing this operation while the patient is in good condition.

Tracheal wounds, if not too extensive, have a tendency to close or may be closed by suture of overlying fascia, muscle or adjacent thyroid tissue.

Wounds to the skin adjacent to openings in the air and food passages are allowed to remain open until it is certain that the wound is free from gross infection. Closure of the wounds may be made after five days by suture or positioning of the head and proper bandaging. The routine use of penicillin appears to be a definite factor in the avoidance of local and spreading infection.

FRIEDBERG, Chicago.

TRACHEOBRONCHIAL TUBERCULOSIS. MANUEL RODRIGUEZ DUTRA, Rev. de otorrinolaring. **8**:24 (March) 1948.

In a survey of cases of pulmonary tuberculosis in the department of bronchopulmonary diseases of the Salvatore Hospital and associated institutions, the author treated 342 patients with bronchoscopy. On the basis of the results, he makes the following conclusions: Routine bronchoscopic examinations are not recommended in cases of pulmonary tuberculosis. Tuberculosis of the trachea and bronchi occurs in 21.6 per cent of cases (the sex distribution is equal), and this form of the disease increases in frequency with the increase in age. There is no appreciable variation in frequency of tracheobronchial tuberculosis with relation to the stage of the pulmonary lesion, but in 68 per cent of the cases the pulmonary lesion exhibited cavity formation. Atelectasis, particularly lobar atelectasis, is due to tracheobronchial tuberculosis. Bronchoscopy alone rarely verifies the diagnosis of tracheobronchial tuberculosis. The author has been using 30 per cent silver nitrate with moderate success, but does not believe this treatment is the solution to the problem. He anticipates that streptomycin and other antibiotics may supplant the present method. Electrocoagulation is useful, but should be used cautiously, since there is a risk of producing a stricture. The diagnosis of tracheobronchitis only occasionally implies stenosis of either the trachea or the bronchi. Collapse therapy should be used simultaneously with the bronchoscopic treatment. Only in the presence of a pronounced stenosis is there contraindication to collapse therapy.

PERSKY, Philadelphia.

Miscellaneous

SOME PRESENT-DAY CONCEPTS OF HEADACHE. JOSEPH H. HERSH, Ann. Otol., Rhin. & Laryng. **56**:98 (March) 1947.

The author analyzes 1,000 cases of headache and discusses the mechanism whereby the pain is produced. Of the 834 patients in the group who had primary conditions as the basis of the complaint, 282 had vasomotor rhinitis and 241 cervical myositis as the cause, and the two conditions were associated in 470 instances. The importance of a thorough study of each case is stressed, as is the need for recognizing that myositis is an active cause. Mechanical obstruction of the nose was the prime factor in 90 cases, sinusitis in 88 and neuropsychiatric disturbances in 75; the rest of the cases were scattered among various causes. All of the conditions named were found in many other cases as accessory factors, and one should recognize that there may be several factors in any given case.

M. V. MILLER, Philadelphia.

HOW PARENTS AND TEACHERS CAN HELP PREVENT STUTTERING IN CHILDREN. LEON LASSERS, Pamphlet, 8 Keys to Normal Speech, 1947, pp. 1-64.

This article presents in popular style the general advice given by most speech clinics to parents, teachers and others, consisting in the expansion of eight general principles of the prevention of stuttering:

1. Do not label your child a "stutterer" or his way of speaking as "stuttering." Accept his speech as a perfectly natural way for him to talk.

2. Do all you can to give your child a feeling of security, confidence and success. Make him feel that he is worthy and loved.

3. Try to make your child's home a calm, pleasant place, free of family quarrels, conflicts and tensions.

4. Give your child a chance to talk under the most favorable circumstances.

5. Try to make your child's school and classroom experiences happy, successful ones—relatively free of pressures and tensions.

6. A child may safely be trained in right-handedness from infancy, but if left-handedness consistently asserts itself or has become fixed, seek guidance of an expert.

7. Keep your child in the best possible physical condition.

8. Give adequate mouth, ear and rhythm stimulation. Use daily rhythm, rhyming and listening activities, sound play, speech games and conversation.

PALMER, Wichita, Kan.

News and Comment

GENERAL NEWS

DR. ALFRED LEWY RECEIVES AWARD

At the meeting of the Chicago Laryngological and Otological Society on November 1, Dr. Alfred Lewy was presented with the traditional cane of the society, which was started in circulation by the late Dr. Joseph C. Beck. It is the temporary property of the senior living member of the society. It contains in its handle models of several otolaryngologic instruments.

In accepting the presentation, Dr. Lewy thanked the members of the society and stated that he felt that he had earned it by a wise choice of ancestors; he said that he intended to hold on to it and not to pass it on to any successor for a long time to come. The cane has been in the possession of the late Drs. Joseph C. Beck, T. Melville Hardie and George Shambaugh Sr.

EXAMINATIONS

The American Board of Otolaryngology will conduct examinations in 1949 on the following dates: May 11-14, Biltmore Hotel, New York; October 4-7, Palmer House, Chicago.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

At the eleventh meeting of the Central Illinois Society of Ophthalmology and Otolaryngology, held in Springfield November 12 to 14, inclusive, the following officers were elected: president, Clifton S. Turner, M.D., Peoria, Ill.; president elect, Harold Watkins, M.D., Bloomington, Ill.; vice president, Meredith Ostrom, M.D., Rock Island, Ill.; secretary-treasurer, Philip R. McGrath, M.D., Peoria, Ill.

Book Reviews

The Rehabilitation of Speech: A Textbook of Diagnostic and Corrective Procedures Based on a Critical Study of Speech Disorders. Book One: The Pathology of Speech and the Rationale of Its Rehabilitation. By Robert West. Edition 2. Book Two: Remedial Principles. By Lou Kennedy and Anna Carr. Book Three: Case Histories. Price, $5. Pp. 637. New York: Harper & Brothers, 1947.

This is indeed a masterpiece in the field of speech rehabilitation. The authors point out repeatedly the necessity of consulting early and frequently with many specialists if one is to succeed in bringing back normal speech to the handicapped patient.

Careful histories are explored, both for diagnosis and therapy. Prognosis is kept in abeyance, for the sake of parents and patient alike. It is pointed out that many with seemingly hopeless loss of speech improve remarkably under persistent and diligent treatment. Patience is indeed a virtue in the man who aims at being a speech clinician. He must learn to be tactful, gentle but firm, humorous but not fatuous, critical and yet not censorious. Interpretation of physical and mental defects is all important if one is to succeed in this difficult art.

All work should be done in pleasant surroundings, on an individual basis, especially in the case of adults, and strictly by appointment. It is insufficient for the patient to practice only in the clinic; a certain amount of home work is necessary for both child and adult.

The otologist plays an important part in diagnosis and prognosis in those instances in which defects of speech are complicated by varying degrees of deafness. The psychiatrist and the psychologist contribute immeasurably to the rehabilitation of stutterers of all ages. Freudian neuroses play a part in many young girls, and the speech clinician must be equipped to deal with these neuroses. He must be aware of physical and mental handicaps, of personality maladjustments, and must have a diplomatic approach at all times.

Certainly, a well equipped clinic is of value; mirrors, recording and play back machines, toys and games all add to the success of wiping out speech difficulties. However, there is no substitute for the keen observer who is supported by a vast experience in medicine, psychology, psychiatry and speech training. It requires a monument of knowledge and detailed, painstaking study to come out on top in this exceedingly worth while art of speech rehabilitation.

To read this analytic dissertation is to be impressed by the tremendous amount of work required in its compilation. It should be a "must" in the library of every one who is interested in children or in adults afflicted with any speech difficulty whatsoever.

A Handbook of Roentgen Diagnosis of the Skull, Sinuses, and Mastoids. By Barton R. Young. Price, $6.50. Pp. 314. Chicago: The Year Book Publishers, Inc., 1948.

This is indeed one of the finest handbooks of radiology for the specialty of otolaryngology that I have ever seen. The 140 plates are absolutely superb. The delineations and associated captions are extremely well done.

The various technical positions, such as Caldwell's, Waters,' Laws' and Stenvers,' are so clearly explained and shown by actual pictures that they remain engraved on one's memory.

The normal and the abnormal in the sinuses and the mastoid processes are beautifully illustrated by actual case pictures. There is no mistaking the vast amount of work and knowledge that went into the compilation of this specific volume. It should be bought and read, and reread, by every otolaryngologist who professes to do any surgical work whatsoever on the ear, the nose, the throat or the skull.

Diseases of the Nose, Throat and Ear. By W. L. Ballinger, H. C. Ballinger and J. J. Ballinger. Ninth edition, thoroughly revised. Price, $12.50. Pp. 979. Philadelphia: Lea & Febiger, 1947.

This book is indeed encyclopedic in nature. There is little of real importance in otolaryngology that is omitted. Of course, it is impossible to go into the minute detail so necessary in highly specialized operations—for example, all the different types of rhinoplasties. Separate volumes and articles have been written by Fomon and by Lempert from which the graduate student and the specialist can learn minutiae.

A new chapter, "Headaches and Neuralgias of the Face and Head," has been added. Dr. J. D. Kelly has revised his technic of arytenoidectomy for bilateral paralysis of the recurrent laryngeal nerves.

Dr. Gabriel Tucker has added all the new information available on "Peroral Endoscopy."

Many plates and drawings have been added, and they are really excellent. The operations in otolaryngology are described step by step and can be appreciated only by the man who has had to suffer through them by trial and error. No book can be a substitute for actual training at the operating table, nor for the experience gained from many years of treating patients. However, as a fine reference work, in one large, beautifully illustrated volume, one could hardly wish for a better book than that written by the three Ballengers.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Fourth International Congress of Otolaryngology

President: Dr. V. E. Negus, London, England.
General Secretaries: Dr. F. C. W. Capps and Dr. W. A. Mill, 45 Lincoln's Inn Fields, London, W. C. 2.
Place: London. Time: July 18-23, 1949.

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Henry B. Orton, 224 Delavan Ave., Newark, N. J.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Atlantic City, N. J. Time: June 6-10, 1949.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.

American Laryngological Association

President: Dr. Frederick T. Hill, 177 Main St., Waterville, Maine.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Biltmore Hotel, New York. Time: May 16-17, 1949.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

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Southern.—Chairman: Dr. Watt W. Eagle, Duke University, Durham, N. C.
Middle.—Chairman: Dr. Dean M. Lierle, University Hospital, Iowa City.
Western.—Chairman: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.

American Otological Society

President: Dr. Marvin F. Jones, 721 E. 60th St., New York.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Jacob Daley, 104 E. 40th St., New York 16.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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CONTENTS

PAGE

Bacterial Allergy in Relation to Asthma. THOMAS P. O'CONNOR, M.D., CHICAGO 145

Speech Therapy: Experiences with Patients Who Had Undergone Total Laryngectomy. ESTI D. FREUD, NEW YORK 150

Tracheotomy in Poliomyelitis Simplified with New Respirator. R. L. PETERSON, M.D., AND R. C. WARD, M.D., BOISE, IDAHO 156

Effect of Pregnancy on Otosclerosis. HAL WAUGH SMITH, M.D., NEW YORK 159

Vocal Analysis. PAUL J. MOSES, M.D., SAN FRANCISCO 171

Invasion of Ethmoid Sinuses by Adenoma of the Pituitary Gland: Report of a Case. D. F. PROCTOR, M.D., BALTIMORE 187

Role of the Septum in Rhinoplasty. JACQUES W. MALINIAC, M.D., New York 189

Ophthalmic Conditions Referable to Diseases of the Paranasal Sinuses. HENRY M. GOODYEAR, M.D., CINCINNATI 202

Tuberculosis of Ear, Nose and Throat Treated with Streptomycin: A Report of Twelve Cases. JACOB L. GREIF, M.D., AND JAMES W. GOULD, M.D., NEW YORK 209

Lymphoid Eustachian Salpingitis: Its Effect on Tubal Patency; Selective Criteria for Nasopharyngeal Irradiation. J. BROWN FARRIOR, M.D., TAMPA, FLA. 221

Unusual Pulmonary Complications Resulting from Prolonged Lodging of Nonopaque Foreign Body in Left Main Stem Bronchus. ARTHUR Q. PENTA, M.D., SCHENECTADY, N. Y. 233

Induction of Anesthesia with Thiopental Sodium in Tonsillectomy. J. K. M. DICKIE, M.D., F.R.C.S.E., OTTAWA, CANADA 238

Solitary Myeloma Mistaken for Frontal Sinusitis. WALTER D. CHASE, M.D., BETHLEHEM, PA. 244

CASE REPORTS:

Perforating Burn of the Tympanic Membrane. JOSEPH W. PUERNER, M.D., NIAGARA FALLS, N. Y. 249

ABSTRACTS FROM CURRENT LITERATURE 252

NEWS AND COMMENT 259

BOOK REVIEWS 260

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES 262